ARCHIVES OF OPHTHALMOLOGY.

A MODIFIED MOTAIS OPERATION FOR BLEPHAROPTOSIS.[1]

By Dr. DANIEL B. KIRBY, New York.

(With three illustrations on Text-Plate XVII. and three in the text.)

THE matter of the correction of ptosis or drooping of the upper lid is one that has stimulated the ingenuity and tested the skill of ophthalmic surgeons.

Many and various procedures have been developed. These may all be found to fall within one of four classes:

I. Removal of thickened tissue.
II. Resection of the tarsus or levator or both.
III. Vicarious use of the frontalis muscle.
IV. Transplantation of the superior rectus muscle.

In 1897, Motais published his monograph on the subject of the transplantation of a tongue of the superior rectus muscle to the upper lid for the correction of ptosis. The classic operation was performed entirely through conjunctival incisions.

In 1907, Shoemaker devised a modification of the original technique by adding a skin incision so that the muscle tongue could be accurately anchored to the tarsus in the proper position. This added much to the popularity and success, of the operation in this country.

[1] Read before Eye Section, New York Academy of Medicine, January 16, 1928.

In 1924, while studying on the cadaver, the surgical anatomy of the parts concerned in this technique, I discovered that the entire operation could well be done through the skin incision alone. This procedure eliminated the conjunctival

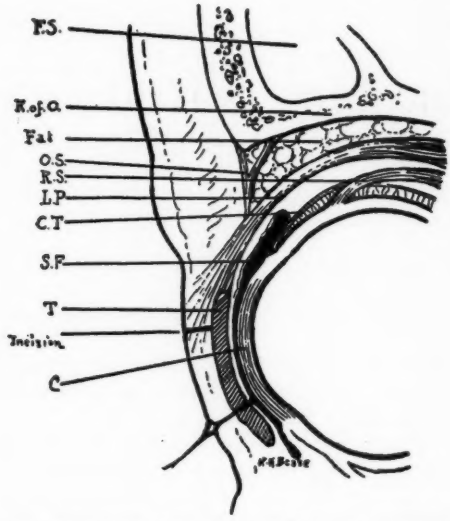

FIG. 1. To show surgical anatomy of the parts concerned in ptosis operation. Sagittal section. F.S. = Frontal Sinus. R. of O. = Roof of orbit. O.S. = Orbital Septum. R.S. = Superior Rectus Muscle. L.P. = Levator Muscle. C.T. = Tenon's Capsule. S.F. = Superior fornix of conjunctiva. T. = Tarsus. C. = Cornea.

incisions and sutures and gave all the advantages of the open operation. In the year 1927, the operation was performed twelve times with good clinical results.[1]

Briefly, the technique at the present stage is as follows: Local or general anesthesia may be used. A horizontal incision 25*mm* long is made through the skin and orbicularis to the tarsus, 8*mm* above the lid margin. The aponeurosis

[1] Acknowledgment is gratefully made of the fact that the technique of this operation was developed by the author in the clinics of Dr. John M. Wheeler at the New York Eye and Ear Infirmary and Bellevue Hospital, during the period when the author held the position of Research Fellow for the American Academy of Ophthalmology and Oto-Laryngology.

ILLUSTRATING DR. DANIEL B. KIRBY'S ARTICLE, "A MODIFIED MOTALS OPERATION FOR BLEPHAROPTOSIS."

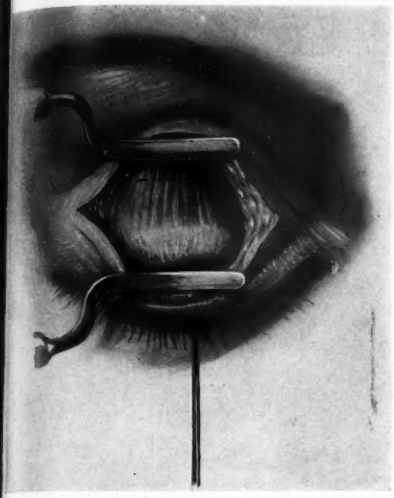

FIG. 2. Shows skin incision which more recently has been placed just below the upper border of the tarsus. The dotted line shows the site of the incision through the levator muscle.

...3. The levator and the smooth muscle of the upper ...re been incised exposing Tenon's capsule covering the ...ior rectus muscle.

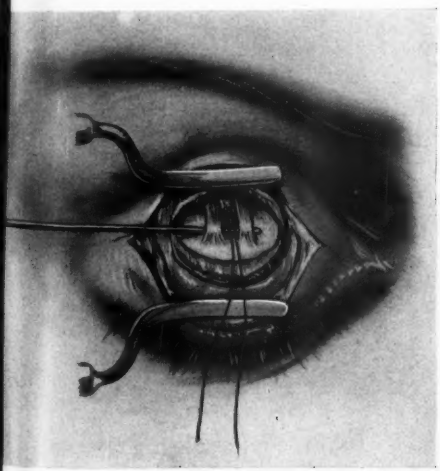

FIG. 4. Exposure of the superior rectus muscle with probe beneath central tongue of muscle fashioned and suture placed.

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of the levator is followed backwards to 8*mm* above the tarsal border. Here a horizontal incision is made through the levator and smooth muscle of the upper lid exposing Tenon's capsule. The conjunctiva of the fornix is held down out of the way by a retraction suture. An opening is made

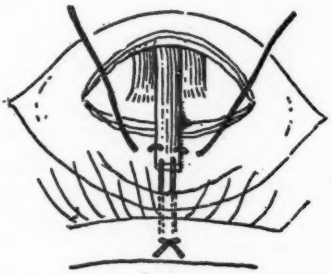

FIG. 5. Shows arrangement of sutures. The primary suture pierces the margin. The supporting suture anchors the muscle tongue to the epitarsal tissue.

in Tenon's capsule at the temporal side of the superior rectus muscle and a hook is slipped beneath the muscle. The latter is exposed so that a tongue of muscle and tendon 4*mm* wide by 10*mm* long can be fashioned. A double armed silk suture is passed through 2*mm* from its end. The muscle slip is transplanted to a pouch which is prepared for it anterior to the tarsus. The needles are passed downwards piercing the lid margin, just nasal to the center of the cornea and the suture tied drawing the muscle tongue and lifting the lid to the position which is required to get the desired effect.

A second suture is used to support the first and to secure accurate and firm apposition to the tarsus. The levator is not necessarily reunited. Excess tissue may be resected. The skin incision is closed by interrupted sutures. A protective dressing is applied. Sutures are removed in 5 to 7 days.

DISCUSSION.

The purpose of this presentation has not been to compare the relative merits of the Motais type of operation with any of the other methods for correcting ptosis, but merely to establish the fact that the transplantation of the superior

rectus muscle to the upper lid can be accomplished entirely through a skin incision and that the method is feasible.

The original Motais operation and the Shoemaker modification are difficult operations. This new operation is not less

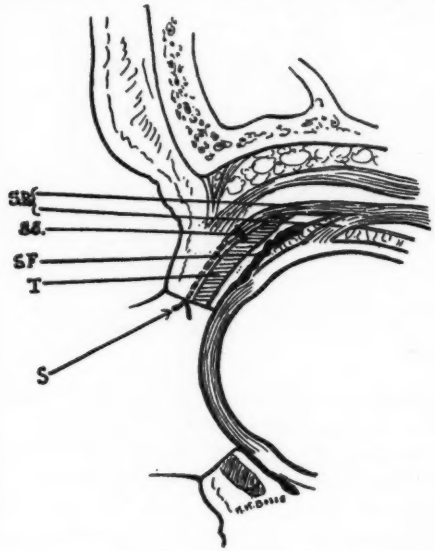

FIG. 6. Sagittal section showing position of tissues after operation. The superior rectus tongue lies in close apposition to the anterior face of the tarsus. S.R. = Superior Rectus. S.S. = Supporting Suture for better apposition. S.F. = Superior Fornix. T. = Tarsus. S. = Primary Suture tied after piercing the lid margin.

difficult. Perhaps, it is more difficult. There is no great danger to the eye, however, but there is need for more accurate knowledge of the surgical anatomy of the parts concerned, and the skill of the operator must be advanced to meet the situation.

RESULTS AND CONCLUSIONS.

The method has been demonstrated to be feasible many times on the cadaver and has produced good clinical results in twelve cases. These cases need not be reported in detail.

Seven of them were operated upon by Dr. John M. Wheeler, one by Dr. I. Goldstein and four by the author. Overcorrection was obtained in one, one was a failure, undercorrection was had in two, while successful results were had in eight cases with satisfaction to the patients. It may be possible to work out a perfect graduation of the effect.

After a successful operation, the patient is able to open the eye naturally, the lid follows the globe in elevation and depression. The lids may be closed voluntarily although there may be a narrow palpebral fissure open during sleep.

There is no permanent deformity of the lid margin, nor is there any permanent impairment of elevation of the globe. The lid in elevation goes back naturally and not directly upwards. There is a tendency for the formation of a fold or crease at the site of the incision, thus making the skin incision an advantage rather than otherwise.

REFERENCES.

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2. SHOEMAKER, W. T. "Observations on the Motais operation for ptosis. Report of three cases." *Annals of Ophth.*, Oct. 1907.

THE AMES SPECTACLE DEVICE FOR THE TREATMENT OF CYCLOPHORIA, WITH A REPORT OF A SUCCESSFUL CASE.

By Dr. WALTER B. LANCASTER, Boston, Mass.

(With one illustration on Text-Plate XVIII. and one in the text.)

THIS case is reported to call attention to a spectacle device constructed on an entirely novel principle which proved remarkably effective in relieving the patient's serious disability; and to call attention also to the work being done on the physiology of vision by Prof. Adelbert Ames, who invented this device.

The patient, a well-to-do professional man of 47 years, had been unable to read as much as ten minutes without causing headache so severe as to totally incapacitate him. In spite of this severe disability at the near, there was practically no complaint as to distant vision. In fact, the patient could read print projected on a screen with a lantern or watch a moving picture without serious discomfort. This condition had lasted for five years or more. He had had the benefit of the best medical and ophthalmic advice and his general health was extremely good. The various ophthalmologists who had treated him were in substantial agreement as to their findings: Normal fundi and media; a small refractive error

O.D. $+ 1.00 \bigcirc + .25$ ax 80 vision $\frac{6}{6}$
O.S. $+ 1.00 \bigcirc + .25$ ax 100 vision $\frac{6}{6}$

2^{Δ} esophoria far, and 5^{Δ} exophoria near; no hyperphoria; accommodation was 1.50 D, much below normal for his age. Prism divergence 4^{Δ}, up and down $1\frac{1}{2}^{\Delta}$ each; near point of convergence 65*mm*. Tests with a Maddox rod before each eye, one horizontal and one vertical, showed that when he was told to place them so as to make a right angle, one line

vertical and the other horizontal, he always selected a position of positive cyclophoria; *i.e.*, a position which showed that his eyes tended to rotate (and when binocular vision was prevented did rotate) so that the vertical meridians tilted outward and the horizontal meridians tilted with their temporal ends down, their nasal ends up. This deviation was not large as I estimated it in this crude way, certainly less than 5°.

Of the various measures which I tried none gave him any considerable relief. He was able at times to read three-quarters of an hour, but after a few days he would break down again and have several days of headache. Then he would start again at ten minutes and gradually increase. (Dyer's method.)

Trying to read with one eye covered was not persisted in by the patient although strongly urged by me. As is so often the case with this treatment, the initial stages are accompanied with such annoying symptoms, and in his case with so much headache, that it is seldom a patient persists long enough to acquire a mastery of the method and an ability to get the benefit which it undoubtedly gives to some patients in whom the mechanism for maintaining binocular vision is out of order.[1]

At first it seemed to me that the combination of very deficient accommodation with inability to do near work but *without trouble at the distance* meant that by giving the patient glasses to do his accommodating and adding prisms to produce a suitable muscle balance would surely enable him to read. Why not? He could read a book if the pages were projected by a stereopticon on a screen across the room or even as near as ten feet; surely if the print were moved nearer and at the same time convex lenses were used to make accommodation unnecessary and prisms to make convergence unnecessary, (say 2.50 added to his distance correction with prisms base in to correct the exophoria caused by moving the print to a point 40*cm* from the eye), under these conditions he ought to be able to read with comfort.

[1] Please notice that I am careful not to say "patients who have some weak muscle." The trouble is not with the muscles but with the mechanism of coördination.

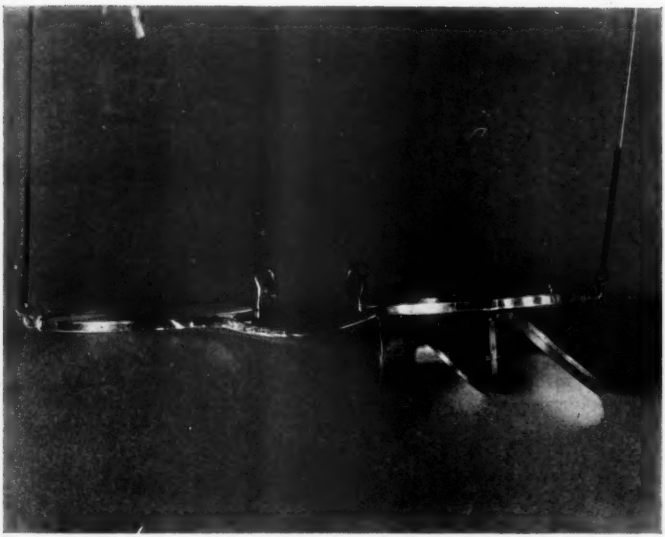
To be sure, there was some relief reported at first, but it soon became apparent that the treatment was a failure so far as enabling the patient to read freely was concerned. It looked as though we should be obliged to fall back on the unsatisfactory diagnosis of hysteria to cover our ignorance.

About this time, it was suggested that the patient permit Prof. Ames to make a thorough study of his eyes from a physiological point of view. Of course I heartily approved—in fact already had discussed the case with Prof. Ames.

After several days of intensive study of the patient's eyes and visual physiology Prof. Ames sent me a very full report of over twenty pages of manuscript besides many charts and graphs. He confirmed the findings of the ophthalmologists as to refraction and horizontal and vertical phoria and found even less accommodation than I had recorded. The chief interest is in the cyclophoria findings. Prof. Ames made his measurements on a modification of the apparatus described in his paper on cyclophoria published in the *Journal of Physiological Optics* of the American Optical Company a few years ago. It is a method of using Volkmann's discs with various refinements and precautions. Prof. Ames will doubtless soon publish more information about his methods and results. He found that this patient showed a positive cyclophoria (declination of vertical meridians toward temporal side) in each eye. This amounted to about 2°. He found that his cyclofusional amplitude was normal in amount but was displaced in the direction of his cyclophoria; *i.e.* outward. This cyclofusional amplitude corresponds to amplitude of convergence and divergence. Thus a patient with 10$^{\Delta}$ of exophoria may have a divergence[1] of 14$^{\Delta}$ (2 meter angles)

[1] This is often erroneously called power of abduction. Abduction is the power of either eye to rotate toward the temple and is measured on the tropometer or similar instrument. It is not the measure of the strength of the abducens muscle but is a measure of the limit of rotation outward of which the eye is capable depending on various factors, check ligaments, etc. Divergence is a *binocular* matter and is measured either by prisms base in or by an instrument like an amblyoscope or like the instrument used by Prof. Ames which has an object for each eye and is so constructed that the angle of the visual axes can be measured as they diverge with separation of the arms of the instrument. Divergence is a binocular matter, and so is convergence, but a one-eyed man has abduction and adduction.

ILLUSTRATING DR. WALTER B. LANCASTER'S ARTICLE, "THE AMES SPECTACLE DEVICE FOR THE TREATMENT OF CYCLOPHORIA WITH A REPORT OF A SUCCESSFUL CASE."



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measured with prisms—he can maintain single vision (fusion) as prisms base in before the eyes are increased up to 14$^{\Delta}$, beyond that he can no longer fuse the images of the two eyes and sees double. He may have a near point of convergence of 125*mm.* = 8 M A. Then his amplitude of convergence is 2 + 8 or 10 M A, of which 2 is negative convergence (divergence) and 8 is positive convergence.

In a similar way the eyes have the power of fusing images which are displaced not in or out as in convergence and divergence but torsionally, rotating around the axis of the line of vision (negative or positive declination). This amplitude of cyclofusion amounts to several degrees each side of the zero point. There are, of course, various precautions which have to be taken in making these measurements, the most important being not to use vertical lines for the test object since when rotated away from the vertical they can be fused in two ways (a) by perspective (third dimension) and (b) by torsion of the eyeball. (a) is much greater than (b) and so might easily obscure it and cause error. In a normal person the cyclofusional amplitude is divided so that one can fuse objects rotated in the positive direction or in the negative (nasal) direction about equally well.

In the case of this patient the power to fuse or overcome torsion of the fields outward (temporal declination) was abnormally large, 4$^{\Delta}$, but the power to overcome torsion of the fields by torsion of the eyeballs inward (nasal declination) was almost zero and even at times when tested at the near point and after fatigue was less than zero; *i.e.*, there was cyclotropia.

It seemed clear that this was an important factor in causing the patient's symptoms. The range of cyclofusion when looking at distant objects included enough negative torsional power to fuse the images of the two eyes but at the near the lesser amount (and at times total lack) of negative torsional power made fusion too difficult to maintain. Why was the cyclofusional amplitude greater on the negative side in distant vision than at near vision? Possibly it was due to the influence of looking more downward and trying to converge and accommodate. We do not know enough about this function to be positive.

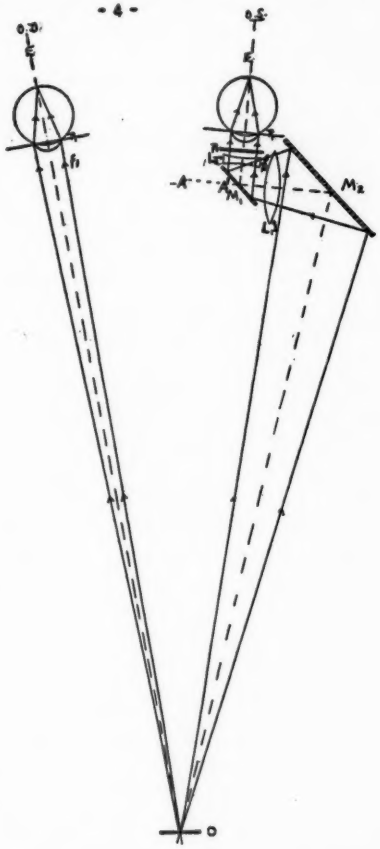
Heretofore in the treatment of cyclophoria we have been limited to covering one eye or operating. The use of oblique cylinders may be mentioned only to condemn it. To be sure, an oblique cylinder will tilt a vertical line to the right or to the left according to the direction of the axis of the cylinder, but at the same time it tilts horizontal lines in the opposite direction and does not tilt at all lines parallel to its axis or at right angles to its axis. In other words, it does not rotate the field, it only distorts the field and therefore introduces a factor which in itself often causes eyestrain.

To give relief to this patient, Prof. Ames invented a new device based on a principle hitherto unused in spectacles. The following experiment will make clear the principle involved.

Hold a hand mirror vertically before you but at an angle of about 45° with the line of vision and a few inches from the eye. You will see in the mirror an image of the wall of the room on your right or left according to which way the mirror faces. If the mirror is held vertically, then vertical and horizontal lines on the wall (edge of door or shelf) will appear vertical and horizontal in the mirror. Now tilt the mirror so that it is no longer vertical. Two things happen. The image is moved up or down (as it would be by looking through a prism base down or up) and *it is tilted or rotated to the right or left* (cyclotorsion) so that vertical and horizontal lines are tilted.

Since we need to produce tilting of the field of vision in front so that we can look at objects in front of us, we must use two mirrors, one being stationary and used only to change the direction, not to do any of the tilting. Hold two hand mirrors, one in each hand, in the positions shown in the illustrations, M_1 and M_2 so that the image of the wall before you is reflected from one to the other, and then to the eye. When the mirrors are held vertically, so that you can see a vertical line, say the edge of a door frame, in its proper position, you will find that on tilting one mirror toward the other so that they are no longer parallel and vertical, you do two things to the reflected image of the vertical line: you move it up or down according to the way you tip the mirror, and you tilt the vertical line (and the whole image of the wall, of course)

to the right or left—cyclotorsion. If you wish to produce torsion only, without the vertical displacement, you can use a suitable prism to neutralize the vertical displacement for



any given rotation. This has been reduced to practical dimensions, so that it can be worn comfortably. It is provided with screws, etc., so that adjustments can be made for any degree of cyclophoria, with lenses for any refractive error and prisms or tilting for any vertical or horizontal phoria.

The patient obtained complete relief from the first day and has been using this device for his reading for over a year with the greatest satisfaction.

The following technical drawing and description are by Dr. Gliddon, assistant to Prof. Ames, who made the optical calculations required.

The path of the rays is from the field through the rotating device to the eye E.

The rotating device consists of two mirrors M_1 and M_2 parallel to each other and at 45° to the line of sight. By rotating the mirror M_1 about its axis AA′ the field of view O is rotated. At the same time the field is displaced. To counteract this displacement a prism R is placed at R. Any desired amount of rotation in either direction can be obtained by rotating the mirror M_1 about the axis AA′ and using the proper prism R base up or base down. For distant vision this is all that is necessary.

For near vision when the field is say 30 to 40*cm* from the eyes the image seen by the eye looking through the device is noticeably smaller than the image seen by the other eye, due to the fact that the path of the rays coming through the device is longer. To counteract this and make the images in both eyes the same size, lenses L_1 and L_2 are introduced. Their effect figuratively speaking is to carry the nodal point of the eye forward and thus to increase the size of the image. The power and position of these lenses relative to the eye has to be proper for a particular distance of the object field O.[1]

The desired convergence for near vision is obtained by tipping the mirror M_2 about a vertical axis.

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[1] If the object O is 40*cm* from P^1 (first principal point) of O.D., then the path of light to O.D is 400*mm*. Because of the mirrors, etc., before O.S. the optical path in O.S. is 432.3*mm*. This makes the image in O.S. only 0.945 times the size of the image in O.D., so it is necessary to put a + 3.25 D (L_1) sphere between the mirrors (36*mm* from P or 19*mm* from F of eye) and a —3.25 D sphere (L_2) at f_1 of the eye. This increases the size of the image in O.S. so that both images are the same size.

EYE-STRAIN IN RELATION TO FUNCTIONAL NEUROSIS.

By Dr. F. W. MARLOW, Syracuse, N. Y.

(With one chart in the text.)

THE term psychoneurosis is of comparatively modern origin, and is defined as "a disturbance of some function or functions in a healthy person not depending upon some physical condition." In order therefore to make a diagnosis of a psychoneurosis it is necessary to eliminate every possible physical condition that may account for it. As it is rarely true that we are able to do this with certainty, it is equally rarely true that a diagnosis of psychoneurosis can be made with certainty.

Ross, in his work on functional nervous disorders reviews the various etiological theories in vogue at different periods for the last fifty years or so and shows how each has been discarded in turn. The Weir Mitchell treatment, to which a great many successes were attributed in the past, is no longer efficient, the difference being due to the changed mental attitude of the doctor towards it.

In turn the importance of displaced uteri, floating kidneys, etc., has loomed up and faded away.

Similarly with eye-strain. According to this author, the day has passed when much importance is attributed to this group of causes as generative of functional nervous disturbance.

In a recent paper read before the ophthalmic section of the A. M. A., Lunt and Riggs emphasized the fact that in all illnesses the psychoneurotic element is a very important factor and that in ophthalmic practice 25 to 30 per cent. of the cases

are of this origin. How this percentage is arrived at is not apparent. That, however, is not the point upon which stress should be laid. What it is desirable to emphasize as strongly as possible is that in many cases the ocular examination is not complete enough to justify any opinion as to the symptoms being of ocular origin or not.

In the majority of cases an opinion seems to be based on an examination of the refraction only, often without cycloplegia.

In a certain number an examination also of the muscle balance and the factors relative to it, is made.

Very rarely is any investigation made as to the possibility of a latent fault in the muscle balance, latent in the same sense that hypermetropia may be latent.

Such latent errors cannot be detected by the ordinary methods in use, and as no myoplegic, analogous to the cycloplegics, is available for relaxing the extrinsic muscles, resort must be had to prolonged occlusion of one eye, making all attempts at binocular fusion impossible, for a sufficient length of time to permit the spasm of the extrinsic muscles to pass away, and any deviating tendency to show itself. When this is done, etiological factors may be revealed which are entirely adequate to account for any neurasthenic or psychoneurotic symptom.

If it be admitted that a latent deviation, whether hyperphoria, exophoria or esophoria, or a combination of these, entails an undue expenditure of nervous energy and may therefore induce a condition of nervous exhaustion,—and the evidence of this is unlimited,—then it must be admitted that before the eyes are acquitted of responsibility for its production, it is essential that the absence of such conditions shall be shown as conclusively as possible; and, to repeat, this cannot be done without an adequately carried out occlusion test.

The case here recorded seems to demonstrate very conclusively the importance of highly latent imbalance and the value of this method. It is notable on account of the long duration and severity of the symptoms, and the fact that the patient was examined and prescribed for by a number of men of national reputation with repeated and final failure to obtain relief. No one of them carried out an occlusion test,

though one gave the opinion that there was a lack of co-ördination of the extrinsic muscles.

The following is mainly the patient's own account of his case.

G. M. Instructor in psychology. "Born 1895. No significant trouble with eyes during childhood except some photophobia while snow was on the ground, 1902, and some trouble while reading by lamplight, 1905 to 1907.

"Used eyes a great deal 1908 onwards. No serious trouble till 1911, when strain during reading (either by artificial light or sunlight) was frequent enough to be very troublesome. Visited Dr. S., who prescribed glasses for reading only. (R −0.50C 80°, L −0.62C 90°). Used these (and shortly thereafter constant-use glasses) (R −0.75C 100°, L −0.62C 75°) with tolerable success until late in 1912. Eye-strain became frequent in 1912–3, and in April, 1913, eyes practically collapsed; could only use them with intense discomfort and much effort. During this time and thereafter Dr. S. referred to the trouble as *conjunctivitis*, and applied an alum stick to the lids. In October, 1913, eyes being very bad, went to Dr. A., who told me to leave off glasses altogether (and prescribed zinc sulphate drops). Suffered greatly from constant strain, and was obliged to leave much of my work undone. In February, 1914, succeeded in getting Dr. A. to prescribe glasses, (R −0.12C 90°, L −0.25 C 80°) but these were so weak as to be of very little use. In April went to see Dr. C., who dilated the pupils (the first who had done so) and told me 'You have three times as much astigmatism as your glass corrects.' Accordingly he gave me glasses (R −0.75C 100°, L −0.50 C 65°) which were much stronger (and in fact closely resembled those given by Dr. S.). He also prescribed sodium biborate drops. Eyes responded immediately and profoundly, and for the next few years I enjoyed almost unlimited use of my eyes without serious strain. Whether the months without glasses or with weak glasses had in some way made the eyes more susceptible to correction, or whether the slight difference between Dr. S.'s and Dr. C.'s glasses was important, or whether the change of eyedrops was the significant thing, I am, of course, unable to tell. *At the time*, I attributed much importance to the soothing effect of the drops, but I discontinued their use after a year or so without my eyes being any the worse. Between the condition of my eyes before and after seeing Dr. C., there was at any rate an immense difference. One fact during this time is perhaps of special interest. In 1915, while doing some hard studying, my eyes suddenly

went bad. I tried the experiment of putting the hand over one eye while I worked with the other, alternating them as they became fatigued, and discovered to my great surprise that I could in this way keep going for a couple of hours. After a few weeks of this, my eyes returned to fairly satisfactory binocular use. In 1917 I again had some trouble and went again to Dr. C. He again dilated the pupils, but decided not to make any important change in the prescription. He suggested, however, that my trouble was very likely due to a nasal infection, and advised me to see a rhinologist. I did so, but was advised against any operation. From 1917–20 I had no serious trouble. Again in the summer of 1920 there was nearly a complete collapse while doing steady eye-work. I visited the Boston Eye and Ear Dispensary, and had a six weeks course of adrenalin shrinkage treatment of the nasal mucous membranes (no result). Some improvement in the autumn of 1920, and during the next few years much variability from month to month; sometimes I could do several hours work a day, sometimes scarcely any.

"The nature of the trouble, throughout this entire period, may be summarized by saying (a) that some photophobia was always present, growing severe after reading, (b) that reading brought on soreness of the lids, and, if persisted in, the pain became acute, (c) that if persisted in still further, the eyeballs began to ache, sometimes with pain in one or more of the external eye-muscles, (d) if in spite of these warnings I continued to read, blurring of vision and sharp occipital headache ensued.

"Between 1920 and 1925 I saw four additional nose-and-throat specialists. One favored a turbinectomy, one an ethmoidecomy, and two opposed all operation. Drainage is undoubtedly poor, but not poor enough to be significant in itself, and I was never convinced that the operation would help my eyes. So it was not performed.

"But to return to the eyes themselves. In 1920 I saw Dr. V., who gave me a prescription (R -1.25 cyl $112\frac{1}{2}°$, L -0.75D cyl $75°$) which in general served pretty well. During 1922, several months severe trouble led me to see Dr. K. After dilating the pupils and making a very thorough examination he said that Dr. V.'s prescription was correct and that no change was indicated. He did, however, prescribe Crookes *B* lenses as a protection against photophobia (Dr. C. had given me Crookes *A*). This did not help as far as I could observe.

"Difficulty continuing, I went several times in August–October, 1924, to Dr. L. He gave me somewhat less correction than I had had. (R $+0.75$ sph $\bigcirc -1.25$ cyl $113°$,

L +0.37 sph ⊂ −0.87 cyl 72° prism ½° base down). He said, however, that the *chief* trouble lay in the failure of the two eyes to work well *together*. He prescribed exercises with prisms. I continued these daily for about six weeks, without observed benefit. He also prescribed prisms for my glasses. The general condition of the eyes remained unchanged. In February, 1925, I saw Dr. I., who removed the prisms given by Dr. L. and said that as far as the astigmatism was concerned, there was no real choice between the prescriptions of Dr. K. and Dr. L.; one was as good as the other, their difference being so slight.

"In March, 1925, following upon overwork and an attack of influenza, I developed a condition of severe nervous fatigue ('nervous breakdown') and needless to say this aggravated the eye-trouble. From the autumn of 1925 onwards I could seldom do more than 15 to 30 minutes work at a time, and not more than an hour and a half or so per day. I had to do practically all my work through dictation and listening to reading-aloud. In the spring of 1926 I saw Dr. D., who said that Dr. I.'s prescription was correct and that the difficulty was probably due to a focal infection. In May, 1926, I saw Dr. G., who said that I was greatly over-corrected and prescribed a much weaker glass. (R −0.50 cyl 110°, L −0.25 cyl 60°). No change; I could scarcely use my eyes at all.

"It will readily be seen that by this time I was completely discouraged, and believed that the eye trouble was not remediable except in so far as improvement in general health would help more or less."

The patient came under my observation on July 23, 1927, during a temporary residence in Syracuse, very skeptical as to the probability of receiving benefit.

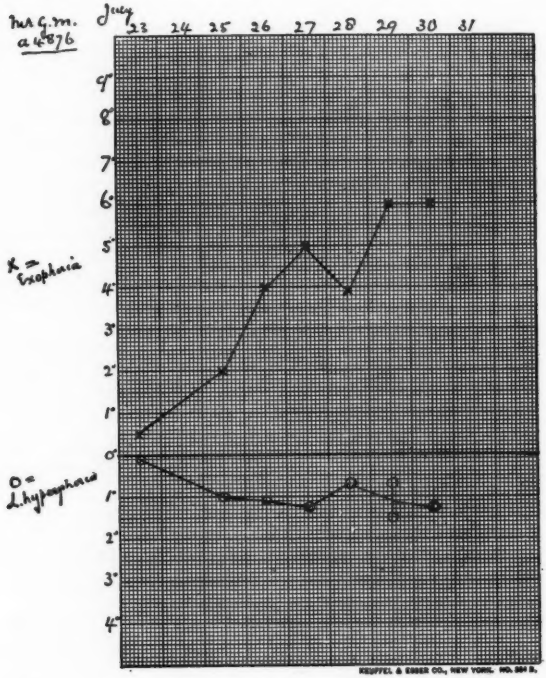
Examination resulted as follows:

The glasses he was wearing were R eye − 50 cyl 110°
L eye − 25 cyl 60°

The right eye now accepted −0.25D or −0.12S ⊂ −1.25C or −1.12C axis 115°. The left eye accepted −0.25 sph ⊂ −0.87C 70°.

Tests for muscle balance showed at 6*m* exophoria less than 1°, left hyperphoria less than ½°, at ⅓*m* exophoria 16° and right hyperphoria ½°. P.P.C. 8*cm*. In view of the previous history of tests with and without cycloplegic, it didn't seem probable that a cycloplegic test would be advantageous and it was omitted. On the other hand the inability to wear with comfort anything approaching a full

correction of his astigmatism, and especially the presence of marked photophobia, his account of improvement when using his eyes separately, and the repeated failure of refractive correction, however varied, to give relief, made it seem most probable that the very pronounced eye-strain, dating from early in life, was due to some latent muscle imbalance. The method of prolonged occlusion was explained to him and readily accepted. The accompanying chart shows the change in the muscle balance, during the week for which the test lasted.



The final test, after seven days' occlusion, showed that he had at least 6° of exophoria and nearly 1½° of left hyperphoria in the primary position. The left hyperphoria was greatest up to the right, and changed to R hyperphoria on looking down to the left. The sursumduction was the same on both sides, 1° to 1½°.

On July 30th he was given the following formula:

R eye −1.12C 115° ⊂ Prism 2° base in up 20°.
L eye −0.12 sph
−0.87C 70° Prism 1¾° in.

"The glasses," continuing his statement, "at first led, if anything, to even worse strain. After two weeks, however, I was reading one morning, shortly after breakfast, and being interested in what I was reading, pushed ahead in spite of some strain. To my surprise I found that no trouble followed; in fact, as I continued, the strain subsided, and I read nearly all day, a feat which I had not accomplished since 1920. The next day I did the same entirely without strain. From that time since, without interruption, I have enjoyed continuous and undisturbed use of my eyes for as many hours as I wish. Some days I have read eight hours, and could have gone on without strain if I had wished. One difficulty remains, a slight photophobia, which hinders, though it does not absolutely prevent reading by artificial light. . . . It is, however, very much less severe than before, blood vessels in the eyeballs are also less conspicuous and pain in lids is much less. . . . There is no doubt whatever that my eyes since August have been better than at any previous time in my life, even times when my general health was much better than it is now. The suddenness and completeness of the change from practically zero efficiency to nearly 100 per cent. efficiency makes me quite confident that at least for my case the prolonged occlusion method was the clue to a very baffling problem."

Cases resembling this one in respect of the difficulty of wearing a full or approximately full correction of the astigmatism raise the question as to the nature of the problem presented. This situation seems to be commonly met by reducing the strength of the cylinder empirically to ascertain how much correction can be tolerated. It would seem that the real problem to be solved is why the patient cannot wear glasses which are an accurate correction of the refractive error. The common explanation will be found to be the existence of a latent muscle imbalance, often demonstrable only by an occlusion test.

A COMPARATIVE STUDY OF THE RÔLE OF PIGMENT IN THE PHYSIOLOGY OF VISION.

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SOME form of pigment can be found in most vertebrate and invertebrate eyes. If one examine a series of eyes representing all the main classes of animals, from the highly developed mammalian eye down to the simple collection of specialized cells in the lowest forms, one or more pigments are nearly always found. This fact becomes more striking the more primitive the type of eye examined. One after another of the more specialized parts may be absent as we descend in the scale until we come to the earliest form of photoreceptor and yet we still find some type of pigment remaining. There are but few exceptions to this rule. Hesse (12) has found unpigmented photoreceptors in Lumbricus and also in some of the leeches. The evidence which shows these cells to be photoreceptors is that the animal reacts more strongly to light in those regions where these cells are most numerous, and that they are similar in their structure to other pigmented photoreceptors. It should be noted, however, that in the various species of worms these cells are imbedded in the epidermis. Further, both these species, leeches and worms living in the ground, must have very little use for more than the merest perception of light. In one other form *i.e.*, Salpen, unpigmented photoreceptors are to be found, according to Beer (2). The question of those higher forms where pigment is abnormally absent, *i.e.*, albinos, will

[1] Read before the Section on Ophthalmology of the College of Physicians of Philadelphia, November 17, 1927.

be discussed later. This constant association of pigment with photoreceptors throughout the animal kingdom suggests that in some way or other pigments are concerned with the act of vision.

A comparative study of pigments in general reveals many interesting facts of possible importance in connection with their function in the mammalian eye.

There are in the human eye at least five types of pigment which may be considered separately:—

1. The Pigment Found in the Stroma of the Choroid and Iris.

This is in the form of small deeply pigmented cells having long slender processes uniting with one another and forming thereby a plexus or meshwork. These cells, known as chromatophores, have no known function, but it is possible that like chromatophores found in the skin of many lower forms of animals they are contractile in nature. This hypothesis was advanced by Muench (20, a) who found that this pigmented network resembled muscle fiber in its finer histological structure. He believed these stroma muscle cells were highly organized and must possess a high degree of contractility. Since he found this network thickest in the region of the blood vessels he suggested that it might serve a vasomotor function, and he pointed out that this might be particularly true in the case of the iris for "it is generally agreed that here the blood vessels lack a muscular coat." In a later paper (20, b) he studied the innervation of these stroma cells and found ganglion cells apparently from the sympathetic nerve in actual contact with this muscle plexus.

Lauber (18) confirmed his findings of the spiral structure of the protoplasm of the stroma cell and its innervation both in man and in *Macacus rhesus*; and later Schock (26) completely confirmed the sympathetic innervation of these cells as being similar to the sympathetic nerve endings described in the intestine by Ramon y Cajal.

Salzmann (24), however, has not been able to see these cross-striations and is puzzled as to what the actual function of such muscle cells in the choroid would be.

The changes which may occur in the skin pattern in the lower animals as a means of protective coloration by contraction of the chromatophores, so-called chromatic adaptation, are beyond the scope of this paper since they have no direct connection with vision save in a few instances; for example, in certain small flat fishes allied to the turbot this adaptation was shown by Sumner (28) to be effected as a reflex through the eyes. When the fish are made blind a change in the background produces no adaptive reaction in the chromatophores. In the majority of animals the reflex occurs through sensory receptors in the skin. In the frog, for instance, the receptors are chiefly in the pads of the toes (Biederman, 3). These are affected by the different physical qualities of the surfaces with which the animal is in contact. In the habitual surroundings of the animal these are associated with definite colors, rough with stone, smooth with grass, etc.

2. The Pigment Epithelium of the Iris.

The pigment epithelium of the iris is not a direct continuation of the retinal pigment layer but represents embryologically the tunica interna (Salzmann, 24) which has here become pigmented and taken on one of the chief functions of pigment in the animal economy, *i.e.*, that of protection against light. The association of pigment to light is an intimate one and takes us somewhat far afield into the subject of the origin of pigments in general.

Pigments are usually formed from a colorless substance chromogen (from tyrosin, a decomposition product of protein) by the activity of oxidizing ferments. These may only occur during certain phases of development, as in the retina of the rabbit embryo they appear about the 12th day and vanish about 8 days later when the pigment has been fully formed, or they may be present throughout life as in the chromatophores of the human skin which may be activated to produce pigment whenever exposed to intense illumination. Hueck (13) has arranged the pigments occurring in the animal body into four main groups on micro-chemical grounds, *i.e.*, hæmosiderin, hæmatoidin, melanin, and lipofuscin. The presence of mel-

anin is apparently limited to the epidermis, the retina and choroid, the ganglion cells of the central and sympathetic nervous systems and the pia mater. Since oxygen is necessary for the formation of pigment it is clear why it occurs chiefly in the outer skin and around canals which contain oxygenated water or body fluids; for example, in vertebrates pigments may be found along the course of blood vessels since the erythrocytes carry oxygen. The fact, however, that white and colored spots of the most varied pattern exist in the skin alongside of each other shows that the cause of pigment formation must be complex.

Another important factor in the production of pigment is light itself. The majority of animals are more deeply colored on the dorsal aspect of the body which is exposed to light. In the same manner snails and mussels are unpigmented in the region covered by the shell whereas the remaining portion is deeply pigmented. The human races living under intense illumination are in general more deeply pigmented than others and the color of birds, fishes, butterflies and insects is much more brilliant in the tropical sun than that of their Northern relatives, who are often somber-hued. It is evident from these examples that light favors the occurrence of pigment and leads to fixed inherited colors. It is not possible, however, except in rare instances to change the characteristic pigmentation except by breeding.

Pigments may also be formed in nature by the breaking down of protein, for example the brilliant red and gold colors of the leaves in Autumn is caused by the decomposition of protein by the frost; and large amounts of pigment may occur in tissues undergoing pathological processes; as in melanosarcoma, etc. With the multiplication of the malaria plasmodium and other protozoa the so-called rest bodies are seen containing pigment due to protein destruction. Rudimentary organs, for example eyes, often contain large amounts of pigment on this basis.

Augstein (1) has shown that the new formation of pigment in the eye is dependent on the interaction of blood extravisation and pigment epithelial cells. No new-formed pigment will occur unless a trace of the pigment epithelium is present at the site of an extravisation of blood.

As Clarke (6) points out "it is undoubtedly true that one function of pigment is to act as a protection to the underlying tissues against too much radiation but at the same time it insures a maximum absorption of radiant energy in the basal cells of the epidermis and may therefore result in an increased light reaction at this point." As mentioned above, the dark color of the races living under the tropical or arctic sun may in some measure be due to the influence of the light itself and is quite possibly of a protective nature. Hemoglobin protects against the ultra-violet rays since it absorbs them. The marked redness of our skin with sunburn is therefore possibly a protective reaction, since the large number of capillaries dilated with blood protects against the entrance of the ultra-violet rays.

The function of the pigment on the posterior surface of the iris is to provide a light-proof diaphragm for the eye. There is one essential difference, however, between the function of the ocular diaphragm and that provided for cameras. In the case of the latter we have a sensitive plate which has an optimal degree of light intensity for its proper exposure. The sensitivity of this plate cannot be altered and the diaphragm or stop aperture is chosen which will admit of this optimal illumination. In the case of the eye, on the other hand, the retina can adapt itself to changes in light intensity provided sufficient time be given. The iris diaphragm serves the purpose of controlling changes in illumination until this adaptation can occur. If the change in intensity of illumination proceeds slowly enough no change in the size of the pupil will result within a wide range.

3. The Pigment Epithelium of the Pars-Iridica Retinæ.

In the course of development the retinal pigment epithelium in the region of the iris undergoes a very interesting transformation. It becomes the dilator pupillæ, the existence of which was proven physiologically by Langley and Anderson (17) before the actual muscle cells were seen with the microscope. Retzius in 1893 was the first to express the opinion that the dilator muscle might possibly arise from the epithelium of the pars-iridica of the optic vessicle and in 1898

Grynfelt, apparently quite independently, came to the same conclusion. The anatomical studies of Heerfordt (11) have shown that Bruch's membrane is not a true elastic membrane but consists of fibers which are entirely similar to smooth muscle cells without nuclei. This membrane is of epithelial origin.

The investigations of Von Szili, Jr. (29), in the anatomy and embryology of the posterior layers of the iris have shown that the epithelial part of the iris corresponding to the rim of the optic vessicle is composed of two leaves from the beginning and gives rise to the iris pigment and the whole iris musculature. The sphincter pupillæ, therefore, as well as the dilator pupillæ is an epithelial muscle. The sphincter develops in man about the beginning of the 4th month and from the epithelial cells of both layers of the optic vessicle. The dilator develops by the transformation of the anterior layer of epithelial cells in the 7th month. The transition from epithelial cell to muscle cell has gone on to completion in the case of the sphincter muscle, whereas in the dilator the transition has gone on in only part of the cell and the remainder has retained its epithelial character and in part its pigmentation.

Mention will be made later on of the movements of the pigment in the pars optica retinæ under the influence of light. In this connection it is interesting to point out the transition in function from amœboid motion to that of contraction of unstriated muscle proceeding hand in hand with the elaboration of structures. Treacher Collins (7) has offered an ingenious suggestion as to how these muscle fibers come to be developed from cells of the neural epiblast. In the lower animals, such as amphibians and fishes, the iris responds directly to light after all nerve connections with the eyeball have been severed. It is known that the pigmented cells which compose the outer layer of the secondary optic vessicle when stimulated by light manifest amœboid movements. They send out processes which protrude between the rods and cones. These phototactic movements are best produced by light of short wave length suggesting that they are the result of chemical change. In the eyes of fishes these cells are continued directly onto the back of the iris and it is not

surprising to find that they still have phototactic power. Those cells lining the retina receive the stimulus on their inner surface and protrude processes out between the rods and cones. Those lining the back of the iris receive the stimulus on their outer surface which have processes protruding from it constituting the dilator muscle of the iris. These processes do not project forward in the iris stroma but run radially so that when they retract in the dark the pupil dilates. "In the course of evolution the contractile power of the muscle tissue of the iris becomes changed from one of direct action to the stimulus of light, into a reflex act. No contraction of the iris tissue to light takes place in mammals when the iris has been severed from its connections with the central nervous system. It loses its phototactic action but retains its contractile power."

There are a few other examples in nature of the development of unstriated muscle cells from epithelium. In man, Kolliker (15, a) has shown that the smooth muscle around the sweat glands is an epithelial muscle, and Haidenhain and Hertwig have shown the presence of epithelial muscles in coelenterates.

4. The Pigment Epithelium of the Pars Optica Retinæ.

In the pars optica retinæ the pigment gives the characteristic speckled appearance to the fundus. The single layer of hexagonal epithelial cells each contain a round nucleus. Each cell forms a low six-sided prism with pigment filling the inner part of the cell; the outer clear part rests on the glass membrane of the choroid. At the inner surface each cell carries a number of fine processes projecting between the outer limbs of the rods and cones. This is always a single layer of rather simple epithelium and in its simplicity and regularity in many cases is unequalled by any other tissue in the vertebrate body.

This pigment serves in the capacity of isolating the light, *i.e.*, preventing the light which has penetrated the sclera and choroid from reaching the retina. It also prevents the internal reflection of light which would blur the retinal image. It probably serves to protect and isolate optically the rods

during photopic vision (Vision in Bright Light, Parsons, 23) and the cones during scotopic vision (Vision in Dim Light, Parsons). If the eye of a frog be removed in complete dark adaptation, the pigment is gathered in the outermost part of the pigment cells, exposing the rods to any faint illumination which may enter. If the eye be removed in the light, however, the pigment will be found to have wandered into and around the outer limbs of the rods, isolating them from the light while the cones have advanced in front of the advancing pigment. Nakamura (22) asserts that the movement of the retinal pigment plays an important rôle in dark adaptation but the experiments of Takagi (30) on two albino children show that normal dark adaptation occurs even in the absence of retinal pigment. It should be clearly noted that the presence of visual purple is not excluded in these experiments.

It is possible that the pigment epithelium also has a secretory function. Kolmer (16, a) in 1909 showed in the rods and occasionally in the cones in different vertebrates numerous spherical bodies which he believes are secreted by the pigment epithelium. His proof for this is the fact that in the dark-adapted retina the drops and granules are larger and more numerous than in the light-adapted retina, and after instillation of pilocarpine, a drug which stimulates secretion in a number of glands in the body, they are found especially numerous. He believes they are concerned with dark adaptation. In 1925 he (16, b) found similar granules in the eye of a chimpanzee.

It is this form of pigment which is most constantly met with throughout the animal kingdom. One gets the impression from an examination of the eyes of the lowest forms that there is a unit structure for visual function. This unit consists in the combination of a light-sensitive receptor in the form of a rod or cone with a basal pigment. The simplest photoreceptors show this combination which is best illustrated by the visual organ of amphioxus. Here we have two different forms of visual organ:

1. So-called beaker or cup eyes described by Hesse.
2. The dorsal visual cells described by Joseph.

The visual organ of amphioxus is exceedingly interesting

and peculiar, not only because it shows the association of visual cell with pigment cell, but also because these cells lie inside the central nervous system, and on this Bovari (4) has built his hypothesis of the phylogenetic origin of the paired vertebrate eyes. We cannot assume that the development of the mammalian eye has followed directly from that of amphioxus located in the central nervous system to the eye as it exists today, an outgrowth of the primary neural tube, but the association is suggestive.

The chief interest in the pigment epithelium as far as vision is concerned centers about its property of absorbing the rays of light entering the eye. Ever since Kolliker (15, b) advanced his hypothesis that the rods and cones were the essential light-sensitive part of the retina, physiologists have held heated debates as to the exact position in the retina where the primary change in energy from light to that of a nerve impulse occurred.

Müller (21) is frequently quoted as having claimed from his experiments that the rods and cones were the light-sensitive elements. It will be recalled that by means of the change in position of the shadows of the retinal blood vessels, when the light entering the eye was changed in position, Müller was able to calculate how far back of the retinal vessels the light-sensitive elements must be. In the original records of the meeting at which Müller presented his paper, it is clearly stated, according to Zoth (33), as follows, "From the size of the parallax the distance of the vessels from the percipient layer can be calculated and this coincides so well with the direct measurements of the rod and cone layer from the vessels that one must consider this or the next adjacent element as the light percipient element." This shows very clearly that Müller considered the outer nuclear layer or the pigment epithelium were in no ways excluded by his experimental results.

The outer nuclear layer is scarcely considered today as being the primary seat of transformation of the light energy but there remain two chief groups of theories, the neuro-epithelial and the pigment-epithelial theories corresponding to the site where the transformation takes place. A third may be added which takes cognizance of both elements.

Since we are concerned here only with the function of pigment in the physiology of vision, the numerous hypotheses ascribing to the neuro-epithelial layer the entire rôle of transformation of energy will not be considered.

The evidence which points to the neuro-epithelial layer is mainly anatomic, other than the experiments of Müller mentioned above. An excellent review of this is given by Zoth (33). The mass of evidence points to the energy transformation being a photochemical one. This being the case our conceptions of the process must be largely influenced by the known laws of photochemical reactions; and in this respect the law of Draper and Grotthus is of prime importance. This law clearly states that for any light ray to be effective photochemically, it must be absorbed. Only those regions of the spectrum can be photochemically active, therefore, which undergo absorption in the eye. Transparent protoplasm, such as the neuro-epithelial layer of the retina, can hardly be thought of as an effective medium for the initiation of any photochemical reaction, since but a very small amount of the entering light could be absorbed. We have, on the other hand, in the pigment epithelium an ideal absorbent for the whole visible spectrum, *i.e.*, a black pigment which absorbs all of the visible rays. If one leans towards a photochemical hypothesis to explain the transformation of light energy into a nerve impulse, it is almost necessary to conclude that the initial stage of this reaction takes place in the pigment epithelial cells. Zikulenko (32) considers from his experiments that the pigment epithelial cell must be regarded as the elementary visual unit, but an analysis of his experiments is not entirely convincing.

It was mentioned above that a combination of neuro-epithelial and pigment-epithelial hypotheses was conceivable, *i.e.*, one which looked on the pigment as the primary site of transformation of the light energy and the neuro-epithelium (rods and cones) as the end organs stimulated by this photochemical or photoelectric change. The development of such a mixed hypothesis has been due primarily to Schanz (25).

In connection with this it is well known that in detachment of the retina the site of separation takes place at the junction of the rod and cone layer with the pigment epithelium.

When this occurs, vision in the affected area is lost, which shows the necessity of the pigment epithelium for vision. Further, as pointed out by Traquair (31) and others "the resulting scotoma for blue is larger than that for red, so that the red field overlaps the blue, producing a true interlacing of the color fields." Since a defect for blue is indicative of damage to the percipient layers, this shows that the separation of the rod and cone layer from the pigment causes damage to the percipient layer over a larger area than damage to the rest of the retinal layers. Again, it is well known that after reattachment with the pigment layer has taken place, almost full recovery of the field may occur.

One of the arguments which has been brought against the importance of the pigment epithelium is the fact that there are many animals in which pigment is almost wholly lacking, *i.e.*, albinos, and yet such animals are not blind. The fact remains, however, that their central visual acuity is very considerably reduced, and in many the animal is almost helpless in bright light. The periphery of the retina still remains sensitive for vision in dim light, probably because of the presence of rhodopsin. This will be considered more fully later on. The poor vision is best explained in albinos on the absence of an absorbing pigment layer; the fact that they are not totally blind is to be expected since all protoplasm must absorb a minimum quantity of light, even though unpigmented; and as is well known, visual purple is probably a sensitizing pigment for the rods, increasing their sensitivity to light of low intensity.

Even in apparently normal individuals, a partial lack of pigment may give rise to symptoms. Brusselmans (5) has called attention to a type of asthenopia which he regards as not due to any refractive error but to an insufficiency of retinal pigment. The patients are particularly likely to complain of photophobia.

5. Rhodopsin.

Ever since the discovery of this pigment by Boll in 1877 an increasing number of experiments have pointed to the importance of this pigment in the act of vision. It is almost certain

that it is of great importance to the eye during dark adaptation and that in some way or other it is an example of one of the so-called sensitizing pigments. Under ordinary conditions protoplasm is not markedly sensitive to light rays. Those rays from beyond the violet end of the spectrum have a marked destructive action on protoplasm, but the usual visible rays from 800 to 400 μμ have no such effect. There are, however, a number of instances in nature in which tissues can be made sensitive to ordinary illumination by the addition of small quantities of certain pigments. This is exactly analogous to the sensitization of the usual photographic plates. These are insensitive to red rays but by the addition of cyan blue they are made sensitive to red in that the red rays are absorbed, so that pictures having the proper brightness value for yellow and red are made possible. Further, the addition of eosine gives the proper values for green and blue objects (the principle of the orthochromatic plate). On the same principle infusoria, which are entirely insensitive to light, can be made sensitive by the addition of a small amount of a dye such as eosine. They may be made so supersensitive that a short exposure to sunlight is sufficient to kill them, although if kept in the dark they survive. There is a disease of sheep, pigs and oxen which belongs to this category. When these animals eat a great deal of buckwheat, they become light-sensitive. The pigment free areas of the skin develop an erisypelatous inflammation which often leads to serious, if not fatal, illness. The disease does not occur in the deeply pigmented parts of the skin and also does not occur if the animal is kept in the dark or if the non-pigmented regions are covered. Buckwheat contains a pigment which sensitizes the skin to light. According to Janet Clarke (6) "It has been found possible to sensitize to the action of visible light bacteria, protozoa, red blood corpuscles, enzymes, ferments, the various substances concerned in immunity, etc. The sensitizing reactions are specific. Although a great many substances sensitize in vitro only eosine, chlorophyll and certain derivatives of hemoglobin have so far been found effective in vivo and the only markedly effective sensitizer for the higher animals is hematoporphyrin." This substance produces the most marked effects. Hausmann (9) injected white mice

with it and found that 0.002*gm* brought on acute symptoms when the animals were brought into the light. Smaller doses result in a chronic light sensitization for months. It has been suggested that the skin lesions in pellagra indicate a light-sensitive condition in man, and Junius (14) has considered it possible that vernal conjunctivitis is caused by such a photosensitization.

Sellards (27) concludes, however, that on the whole light sensitization plays a very minor rôle in the etiology of disease in man.

The striking similarity between the curve of the absorption of visual purple of different wave lengths and the scotopic luminosity curve; the well-developed dark adaptation of the periphery of the retina where visual purple is most abundant, etc., almost force the suggestion that this is an example of a sensitizing pigment whose function is to increase the sensitivity of the rods to light of low intensity. It probably is also present in the fovea according to Hecht (10). Edridge Green (8) maintains it is secreted by the rods, but is primarily concerned with central vision, acting on the foveal cones; the objective evidence for this, however, seems debatable.

Summary.

Each of the various types of pigment found in the mammalian eye shows analogies in structure and function with the various pigments found elsewhere in the animal kingdom. The functions of light-protection, isolation, and absorption, photodynamic sensitization, and phototactic movements, so frequently met with in pigments in nature, are also seen in the fully developed eye. The pigment cell is an essential component of every photoreceptor.

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THE PROTEINS OF THE LENS AND THEIR CHEMICAL CHANGES IN THE PATHOGENESIS OF SENILE CATARACT.

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(With three figures in the text.)

In recent years, the appearance of various immunological preparations intended for the prevention and treatment of senile cataract, the occasional reports of such cases treated in this way and the various theories advanced from time to time for the explanation of the genesis of senile cataract, have all served to stimulate the interest of the present day ophthalmologist in this condition. For a better understanding of this subject, however, a knowledge of the chemistry of the normal lens is primarily essential, and also some knowledge of the various chemical changes taking place in the proteins of the lens in the development of cataract is desirable.

Prior to the time of Moerner, who published the result of his analysis of the normal lens proteins in 1894 (1), a knowledge of the chemical structure of the lens was anything but uniformly accepted and exact. It remained for this chemist to establish a definite chemical proportion and classification of the proteins comprising the lens substance. As the result of his investigations, Moerner showed first that the total proteins comprised about 35 per cent. of the lens mass and that they consisted of a soluble and an insoluble portion. The former soluble portion existed to the extent of 18 per cent. of the lens mass and made up 52 per cent. of the total proteins; the latter or insoluble portion which hitherto had not been really investigated was found to be present to the extent of 17 per cent. of the lens mass and made up 48 per cent. of the total protein.

Then, analyzing the soluble proteins (52 per cent. of the total), he found it to be comprised of two krystallins and an albumin. The first krystallin he called "α" krystallin and the other "β" krystallin. The "α" krystallin was estimated to be 19.5 per cent. of the total proteins, 37 per cent. of the soluble protein and 6.8 per cent. of the lens mass. The "β" krystallin was found to make up 32 per cent. of the total proteins, 63 per cent. of the soluble portion and 11 per cent. of the lens mass. It will be seen from this that the "β" krystallin predominated over the "α" krystallin by nearly twice the amount.

In a further analysis of the "α" krystallin, Moerner found it to consist of the following proportions:—N—16.68; H—6.94; C—52.83 and sulphur 0.56. This he called the "Sulphur poor" in comparison with the "β" krystallin which he found to contain sulphur 1.27 and which he termed the "sulphur rich" krystallin. The third portion of the soluble protein was the albumin which occurred in the small amount of 1 per cent. Concerning their distribution, Moerner found that the soluble proteins decreased from the outer parts of the lens inward, the "α" krystallin being found mainly externally or cortical, while the "β" krystallin occurred mostly in the more central parts of the lens. The albumin showed no noticeable distribution.

Concerning the insoluble protein which Moerner termed the frame-work protein, his analysis showed this to contain N—16.62; C—53.12; H-6.80 and S—0.79. In its distribution the insoluble protein was found to increase from without inward.

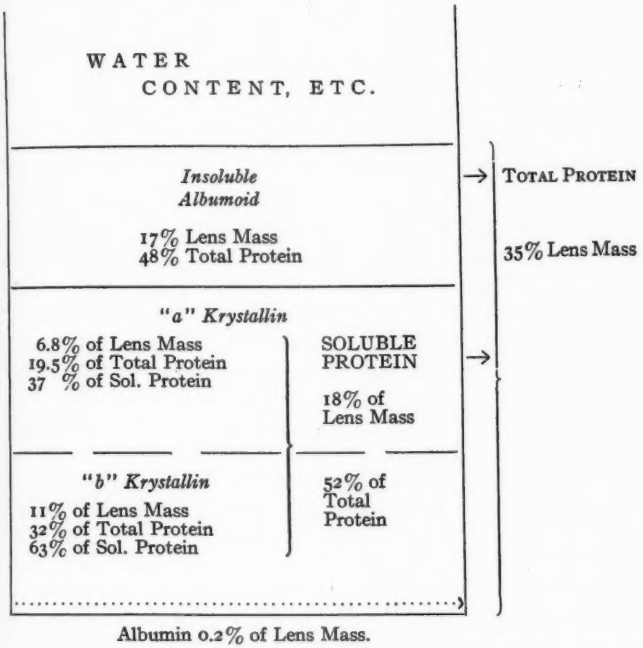
With these detailed results established for the proteins in the normal lens, it is next necessary to turn our attention to a property of lens protein by which there is produced a certain color reaction when treated with 4 per cent. solution of Sodium Nitroprusside and Ammonia. This reaction was fully described by Arnold (2) in 1911, whereby he furnished an important contribution to the knowledge of the chemistry of the lens proteins, of interest in senile cataract.

Arnold found that many proteins, particularly those of the thymus, muscles, blood plasma, mucin of saliva, easein of milk and others, produced a characteristic red color when

treated with a few drops of 4 per cent. Sodium Nitroprusside and Ammonia. These experiments were carried out on animals and it was found that of all the proteins tested, the strongest reaction was produced with the lens protein. The

FIGURE I.

DIAGRAM OF MOERNER'S CLASSIFICATION OF THE LENS PROTEINS.



Albumin 0.2% of Lens Mass.

normal lens, when crushed and treated with a few drops of the 4 per cent. Sodium Nitroprusside and a drop of Ammonia, produced this decided violet red coloring. This reaction of these proteins was said to depend on the presence of Cystein, and was demonstrated in such organs as the liver and others where active metabolism take place, bringing about the presence of free Cystein in those organs. Arnold considered Cystein as an essential constituent of every efficiently functioning cell. It is of great interest to observe the rôle of

Cystein, in the study of this subject, but in doing so we must bear in mind a characteristic which Heffter and Arnold also pointed out, whereby two molecules of Cystein have the ability to unite, with the liberation of their hydrogen atoms, forming Cystine in the presence of Oxygen. In turn, this oxidized product Cystine, in the presence of certain substances like iron, was again reduced back to Cystein. It was not until 1921 that Hopkins discovered that the oxidizable conconstituent in the tissues was a combination of Cystein and Glutanic acid which he called Glutathione.

The practical application of this process of reduction and oxidation will be further considered. Meanwhile, the so-called Nitroprusside reaction of the lens proteins was the basis of further researches by other investigators. Among these, Wiktor Reis in 1912 (3) described the results of this reaction on both animal and human normal and cataractous lenses. Having confirmed the result in the normal animal lens, he found that the normal human lens produced a positive Nitroprusside reaction without any differences in the intensity of the reaction due to early post mortem changes. Also that normally there was no difference in the peripheral and central layers of the lens in the extent of the reaction. But with cataractous lenses he found that the reaction behaved differently in the various stages of senile cataract development. In the mature senile cataract the reaction was negative; in the non-mature form the reaction was present only in the unaffected parts of the lens and depended on the extent of the cataract formation. In traumatic cataract he found the Nitroprusside reaction positive, concluding that in traumatic and in senile cataract different chemical processes must take place.

Reis felt that the reacting Cystein group must be lacking in the lens elements in senile cataract. He could not state for a certainty whether the negative result of the reaction in this condition could be attributed to the deficiency of the Cystein group in the protein of the lens or whether it depended on a reduction in general of the protein contents in cataract, as supposed by Cahn, or was it as Toufesco claimed because of degenerative fatty changes in the lens fibers.

Reis in these experiments gave attention to the whole

In the article "THE PROTEINS OF THE LENS AND THEIR CHEMICAL CHANGES IN THE PATHOGENESIS OF SENILE CATARACT," which appeared in the July issue, the first paragraphs on pages 364 and 369 stated that the reduction to Cystein and Glutathione occurred in the presence of iron, whereas they should have stated that the oxidation of the Cystein and Glutathione was effected in the presence of iron.

lens, showing the differences in the positive Nitroprusside reaction of the entire normal lens and the negative result in cataractous lenses.

It remained for Jess (4) to subsequently apply the Nitroprusside reaction and describe its results with the various fractions of the lens proteins as found by Moerner. In 1913, Jess (4) told how he separated the various soluble and insoluble proteins of the normal lens and tested each separately with Sodium Nitroprusside and Ammonia. He used in this work 120 animal lenses, crushed into a homogenous mass with water and after centrifuging, extracted first, a white percipitate, the albumoid of Moerner. On testing this by the Nitroprusside reaction, it was found to be entirely negative. The insoluble Albumoid, therefore, did not give the characteristic reaction.

From the original solution which produced the insoluble albumoid, he next produced the "α" krystallin by filtering the fluid through kisulgur and transforming the filtrate with 0.1% acetic acid until the precipitate obtained no longer increased. This was then centrifuged and the "α" krystallin obtained tested for the Nitroprusside reaction. A distinct but not very intensive red coloring resulted, showing the "α" krystallin to be positive, but not as strongly as the normal lens substance.

After this the "β" krystallin was separated from the original extract by obtaining a white sediment with alcohol which was filtered and washed. The snow white powder obtained consisted almost exclusively of "β" krystallin. This was found to yield a decidedly positive reaction. These same results were produced on the human lens showing the "α" krystallin to be positive, the "β" krystallin, more decidedly positive and the Albumoid negative.

Jess also confirmed the results of Reis in which he found that cataractous lenses did not produce the Nitroprusside reaction, and attributed their failure to the more or less complete disappearance of the soluble protein of the lens. He stressed the higher Sulphur content in the "β" krystallin as compared with that of the "α" krystallin, his preparation of "β" krystallin yielding the test for Sulphur much more intensively than the "α" krystallin.

After completing these qualitative tests, he turned his attention to a consideration of the quantitative estimates of the separate proteins in the lens and their behavior toward the Nitroprusside reaction. He began by first observing the effect of the Sodium Nitroprusside and Ammonia on lens sections cut with the Mikrotome. In total cataract the color was limited to a very weak red border. In traumatic cataract it was found that the reaction which was formerly found positive, becomes negative in old cases. This modified the result of Reis found in traumatic cases. In one case of 7 months duration and another of 6 years standing, the result was the same as that of senile cataract. In Naphthalene cataract, after long continued naphthalene feeding, with a complete yellowish white dimmed lens, he found a negative reaction in a concentric zone, while in the center of the lens a very small part still colored red. In a still older naphthalene cataract, the central area had also vanished and only in the outer periphery was a very faintly positive reaction perceptible. In his subsequent quantitative estimation of the separate proteins in normal and cataractous lenses Jess (5) carried out a most detailed and thorough examination of their contents. Many difficulties confronted him in this work, particularly in the selection of animals, in whom the determination of the type of cataract is more difficult than in humans. In addition, the ages of all animals were carefully considered, using all the available guides such as teeth, horns, wings, etc. With attention to these details, first a table of weights for the normal lenses of animals up to 16 years of age was established. From this it was seen that the average weights for a pair of lenses ranged from 1.6425 g. and 1.5741 g. in an animal one year old, weight 3 Ztr. (hundredweight), up to about 2.8332 g. and 2.7 g. in one 16 years of age weighing 4 Ztr. The weights were without capsule and it is to be noted that in the comparison between the body weight and the lens weight, the latter is dependent solely on the age. Also that the difference in the weights of a pair of lenses from the same animal was just slightly greater in advanced years than in the younger.

In considering the increase of the weight of the lens in age, Jess found that this was dependent on fixed increases of the

protein content and the water mass with a very slight part being determined by the increase of the ether-soluble substances, but the increase of the total proteins exceeded that of the water so that a change of the proportion took place from 32% protein as compared with 67.5% water, in youth to that of 38% protein compared with 62% water, in older age.

Next the total protein increased with age in normal lenses, yet in advanced age the albumoid content exceeded that of the krystallin, whereas in youth, the krystallin prevailed in mass. The analysis showed that the proportion of krystallin to albumoid changed from 82% K: 18% Albumoid in youth to 41% K: 59% Albumoid in age, or in percentage of lens weight, from 26.46% K: 5.6% A. to 14.88% K: 21.47% A. He next analyzed the content of the ether-soluble elements (fats, cholesterin and lecithin), in the normal lens and estimated it at about 0.30% with a very slight, absolute increase in age but, in proportion to lens weights, not increased. In cataractous lenses, both senile and traumatic, he found the result to be about the same as in the normal, concluding that the genesis of cataract did not depend on an increase of these elements.

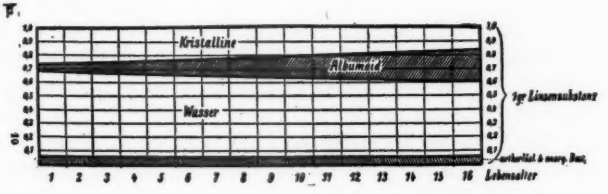


FIG. 2. Change in proportion of proteins in animal lenses from 1 to 16 years of age. (From Jess.)

Now in the senile cataract, there was observed first, a constant decrease of the weights. The absolute total protein content and the absolute water content were lessened constantly, yet the reduction of the water content exceeded that of the total protein. An increase of water, as was assumed in the clinical picture of mature cataract, was never established according to Jess.

Lastly, the decrease of the total proteins followed solely through the loss of the krystallin from the lens, while the Albumoid in the majority of the cases increased.

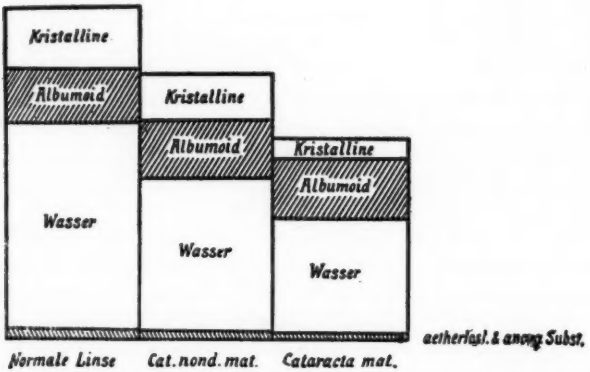


FIG. 3. Showing differences in protein and water content between normal and cataractous lenses, according to Jess.

In traumatic cataract, there was shown in one case no reduction of the weight, a decrease of the krystallins and in most cases also of the albumoids. Never was there an increase of the Albumoid as in Senile Cataract. The relative water content was in some cases increased, and once the absolute water content was found, increased. An increase in fat, cholesterin and lecithin was not established.

By these results he explained the absence of the Nitroprusside reaction to a loss of the cystein-containing krystallin of the lens, which either in part disappears or in part is changed into the increased amount of albumoid, so that no cystein group is contained in its molecule. He concluded that a change into fat of the lens substance did not occur either in senile or in traumatic cataract. The absence of the Nitroprusside reaction noted in the old traumatic cataracts was also traced back to the total resorption of the krystallin.

The question of the disappearance of the cystein with the possible change in the structure of the molecule was the basis for still further investigations. Following Hopkins disclosure (6), that the autoxidizable substance in the cell was a dipeptide

formed by the combination of cystein and glumatic acid which he named Gluthathione, it was shown that this substance had the same oxidation and reduction properties attributed to cystein. Two molecules of the substance combined in the presence of oxygen, liberating the hydrogen atoms. In the presence of iron this was again reduced to its original form. Heffter (7), describing this process with cystein in 1907, stated that the Sulphydril group played a significant part in cell metabolism, since their hydrogen atoms were liberated in the presence of oxygen and then in the presence of other substances later called Catalyzers, they acted as hydrogen receivers, thereby being reduced to their original form.

Since the Nitroprusside reaction was positive with cystein and negative with cystine, the oxidized form, Abderhalden and Wertheim (8), using this reaction as a guide, performed a series of experiments showing that the oxidation of cystein was dependent on its reaction to acid; that pronounced acid reaction retarded oxidation; with litmus neutrality it took place quickly, and in alkaline reaction it occurred quite plainly. Organ extracts producing the Nitroprusside gave the same results.

Investigating the influence of temperature on the oxidation, they found that a cystein solution after two hours at 4 degrees C. gave a 4+ N.P.R.; at 22 degrees C. 3+; at 37 degrees C. 1+ and at 100 degrees C. O. After 4 hours the result of the N.P.R. was at 4 degrees C. 2+; at 22 degrees C. 1+; at 37 degrees C. O.; and at 100 degrees C. O. The influence of temperature was therefore obvious; at 100 degrees the Sodium Nitroprusside reaction vanished after a few minutes.

In studying the influence of metals and light, they found that iron in very small quantities hastens oxidation of the cysteins and copper, mercury and arsenic work in the same way, though not so strongly. Lead, nickel, cobalt, and cadmium were found to retard oxidation. Light was found to have no influence whatever on the process. Even with intense raying with the quartz lamp, no hastening was found.

This influence of light on the cystein reaction of the lens was also another investigation of Jess. Chalapecky (9) had

previously shown that ten pig lenses exposed to the light of a quartz lamp for three hours failed to produce the Sodium Nitroprusside reaction. Jess' (10) tests, however, gave quite different results. He used a quartz lamp, and after numerous attempts, the result was always the same, that the Sodium Nitroprusside reaction after raying the lenses was just as positive as in unrayed lenses, thereby disproving Chalapecky's theory that ultra-violet rays had an influence in reducing soluble protein substances into less soluble modifications.

The influence of Potassium Cyanide on cystein oxidation was also studied by Abderhalden and Wertheim and this was found to retard the process. Organs of animals poisoned with Potassium Cyanide showed the Sodium Nitroprusside reaction more than twice as long as the normal. The obstruction of auto-oxidation by prussic acid was also observed and showed this reaction positive, at room temperature (21 degrees C.) after 12 hours.

The influence of alcohols, chloroform, and ether was found to hasten auto-oxidation of the cystein and also in the tissues, as shown by a weaker or negative reaction with Sodium Nitroprusside and Ammonia. The lens showed this result decidedly.

In the lens, the auto-oxidation was explained as a manifestation of metabolism. In view of the uncertain blood supply of the lens the nutrition is carried on by the numerous lymph channels which, at the same time, carry off the waste products of metabolism. The oxygen provision of the lens followed from adjacent blood vessels, diffusing from them into the lens cells.

Knowing that the two krystallins vanish from the lens dimmed by cataract and that the insoluble Albumoid prevails, Jess (11) next undertook a further analysis of these fractions for the percentage of their structural units according to the more modern classification of proteins as the various amino-acids. He found that the Albumoid was very weak in Alanin (0.8%) as compared to 3.6% in "α" krystallin and 2.6% in "β" krystallin. In Albumoid, Valin was present in traces only, while "α" krystallin showed five times the amount and "β" krystallin ten times. Leucin and its Isomere was obtained in equal amounts (5.2%) in "α" krystallin and Albu-

moid, while in "β" krystallin it was present in half that amount (2.8%). Cystein and Cystine together showed 2.3% in "α" krystallin, 4.9% in "β" krystallin and none in Albumoid. Melanin was 1.1% in "α" krystallin, 0.1% in "β" krystallin and 0.6% in Albumoid. Tryptophan was present in all three. Glykokoll was absent in all three divisions.

No.		*"a" krystallin*	*"b" krystallin*	*Albumoid*
1	Glykokoll	0	0	0
2	Alanin	3.6	2.6	0.8
3	Valin	0.9	2.1	0.2
4	Leucin & Isomere	5.9	2.8	5.3
5	Aspartic Acid	1.2	0.4	6.5
6	Glutanin Acid	3.6	2.7	4.6
7	Tyrosin	3.5	3.7	3.6
8	Prolin	1.8	1.4	1.9
9	Phenylatanin	5.5	4.1	4.6
10	Serin	...	...	...
11	Tryptophan	...	...	...
12	Cystein-Cystine	2.3	4.9	...
13	Ammonia	7.1	11.4	6.0
14	Melanin	1.1	0.1	0.6

Regarding Argynin, Histidin and Lysin, Jess (12) showed that the total quantity of these structural units in the krystallins amounted to about 15% while in the Albumoid almost 17%. Argynin was found in greatest amount in Albumoid (10.26%), while both "α" krystallin and "β" krystallin showed about equal amounts (7.5%). Lysin was slightly higher in "β" krystallin (4.6%) than in "α" krystallin (3.75%). Albumoid showed 3.8% Lysin. Histidin was a little less in "β" krystallin (2.63%) than in "α" krystallin (3.63%), also less in Albumoid (2.74%).

From these results, Jess concluded that, according to the classification of Abderhalden (13), the krystallins should fit with the albumins, rather than the globulins as hitherto, since they contain 15% Arginin, Histidin and Lysin and about 85% single amino-acids; they contain no glykokoll and are soluble in water. The water insoluble albumoid, hitherto classed as a protenoid, similar to Elastin of the elastic tissues, and Keratin of the hair and nails, shows an inclination toward globulin and is so considered, even though it contained no Glykokoll.

These investigations of the differences in the structural units of the separate lens proteins was an advance in the study of this subject. Many explanations for the genesis of cataract had until this time been advanced. One of the earliest, attributing the origin of the condition to the lens itself, was that of Becker (14) in which he held that it is due to an abnormal sclerosing of the lens, whereby in age this process does not proceed completely and with uniformity, thereby hindering a seeping through of the nourishing liquids and causing a sponginess of the layers of the lens next to the nucleus with subsequent contraction of the nucleus. The view of a strictly local cause in the lens was supported by Salus.

Michel and Wagner (15) had examined a number of senile cataractous lenses and determined that the nucleus possessed no protein substance, while in the cortex, traces of Globulin were still found. In other forms of cataract as in traumatic and immature senile varieties, the normal proteins were found.

Leber (16) had found in senile lenses an increase in lipoid content, together with lecithin and cholesterin, and ascribed to these substances an indirect rôle in the formation of cataract. A little later Gross (17) could determine no increase in the lipoid content either in senile or in cataractous lenses. Toufesco (18) declared that the fat content increased with a fatty degeneration of the lens fibres.

Peters (19) maintained that normally an osmotic balance exists between aqueous and lens, which when altered, leads to an interference with the nutrition of the lens and shrinking of the nucleus.

While some of these investigators looked for the cause of cataract in the lens itself, and others desired to attribute it primarily to constitutional changes, Von Hess (20) took an intermediate position and stressed the necessity for observing the various clinical and anatomical behaviors of senile cataract, the continuous and progressive pathological changes in the epithelium of the anterior capsule with subsequent degenerative processes only in the outer cortical layers as in naphthalene and massage cataract. The assumption of deleterious substances entering from without could then be justified. The clinical and anatomical findings in supra- and intranuclear cataract could support the local cause, since here the

capsule epithelium fails and yet the outer layers of the lens show complete normal contents.

Strongly opposed to the strictly local origin of cataract was Roemer, who maintained that the changes in the lens were dependent on primary changes in the blood serum of the individual. Roemer's work (21) covered a wide range of investigations in which he finally turned his attention to the biological condition of the blood serum in senile cataract, arguing that here the blood serum of the individual was different from the normal and that the senile cataract was practically a manifestation of a metabolic disease. Roemer, in his consideration of the subject of cataract and in his experiments, was mindful of the organ specificity of the lens proteins as shown by Uhlenhuth in 1903 (22). He assumed also, that the cells of the lens possessed receptors in the sense of Ehrlich, which receptors were capable of uniting with specific substances for which they had a specific affinity thereby obtaining their nutrition. Certain toxic substances from the blood serum, finding their way into the lens and interfering with its nutrition bring on cataract. In illustration of the relationship of cataract to metabolic disease, he cited Diabetes and in experimenting with the serum of a young diabetic patient with bilateral cataract, he demonstrated specific lens antibodies after injecting the serum intraperitoneally into guinea pigs.

More recently, Guyer and Smith (23), in experimenting with animals, sensitized the serum of fowls with the lens of rabbits, and by injecting this serum into other pregnant rabbits at regular intervals, produced in all the new born rabbits that survived, eye defects which in most cases occurred as opacities in the lens.

In a review of the etiology of cataract, J. Burdon-Cooper (24) stressed the refractive error as having a decided influence on the nutrition of the lens in the production of opacities, and that hypermetropia has a greater influence in this respect than myopia because of the greater accommodation necessary for its correction, and the adverse influence on the ligament and capsule. In discussing the nutrition of the lens, he assumes it is secured by a modified endosmosis of the intraocular fluid; a molecular inbibition and in all probability a

specific affinity of its protoplasm. Disturbance of this nutrition is the initial cause of cataract, the deleterious substances obtaining access to its protoplasm, and resulting in secondary chemical changes, the products of which constitute the opacity.

With regard to the permeability of the lens capsule as a factor in cataract formation, Lowenstein (25) regarded it as a colloidal membrane which, under normal conditions, the electroytes of the aqueous could not penetrate. He considered the alteration in the permeability of the capsule as a possible cause of senile cataract.

With the experiments of Guyer and Smith in mind, and supporting Roemer's view of cataract, A. Edward Davis (26), of New York, had a lens antigen prepared for the treatment of cataract. He first used a human lens antigen in the preparation of anti lens sera, which he administered intravenously, but because of the marked anaphylactic shock, this was discontinued and the subcutaneous injection instituted.

More recently, serologically pure fractional antigens of the lens were produced by Alan C. Woods of Baltimore (27). With "α" krystallin and "β" krystallin this was found possible at their iso-electric point. He also found that they showed the same properties of organ specificity as pointed out by Uhlenhuth in whole lens protein. Another feature was that while the alpha antigen remained stable, the beta antigen after a few weeks became very unstable. It developed a tendency to spontaneous precipitation, as it became older, but when mixed with alpha antigen it lost this tendency to spontaneous precipitation. He concluded that the alpha antigen had an apparent protective colloid action on the spontaneous precipitation of the beta. Wood assumed as plausible that that gradual reduction in the alpha fraction allows the beta to become more pure and thereby removes the protective colloid action. Thus, when the alpha reaches a point where its protective action is entirely removed, the beta krystallin undergoes spontaneous precipitation, with resultant clouding of the lens.

Which again calls to mind the former works of Jess. Since the two krystallins have been shown to vanish from the lens in cataract, he assumed, from the work of Goldschmidt (28),

who demonstrated also the presence of a protelytic ferment in the aqueous, that it is conceivable for these protein bodies, being water soluble, to be broken down by a fermentation process, more quickly than the Albumoid. The cell ferments are liberated with the initial cellular death and go on to decompose the cell further to many fragments. In senile cataract, Hess referred to the early destruction of the epithelium. It can be imagined that from these cells the ferments are liberated which decompose the dead cells and break down the adjacent fibers. The end product would be some form of amino-acids which could pass undisturbed over the lens capsule and be taken up by the intraocular fluid. In nuclear cataract the initial destruction would have to affect some inner lens fibers, followed by the fermentation results on neighboring ones and a dimming of the clear cell substance produced by a breaking down of the krystallins. The cause of the initial cell death, however, as well as the ultimate destination in shifting of the known structural units of the lens proteins remained unsettled. Further investigations concerning the behavior of these structural units, and additional knowledge concerning the process of the chemical changes in the lens proteins accompaning oncoming dimness of the lens, is necessary to further the study of the pathogenesis of senile cataract.

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A CASE OF GLIOMA OF THE OPTIC NERVE.[1]

By Dr. HERBERT WRIGHT WOOTTON, New York City.

(With two illustrations on Text-Plate XIX.)

THE patient, 17 mos., male, was referred by Dr. Wm. J. Hicks of Middletown, N. Y., and was first seen by him June 8, 1927, at which time the left eye deviated outward with marked proptosis. According to the parents, this condition had developed gradually during the preceding three months. Direct reaction of the pupil to light was present. Dr. Hicks saw the case again on September 1st, when all the movements of the eyeball were greatly impaired, external rotation being only about two-thirds of normal. The degree of proptosis had remained about the same, but the direct reaction of the pupil was absent, the consensual reaction being preserved. The disc showed a papillœdema and the eye was blind, vision having apparently been lost during the past two months. There were no evidences of Recklinghausen's disease, and an X-ray showed no enlargement of the optic foramen.

On September 5th, I performed a Krönlein operation and found that the optic nerve, from a point about two millimeters behind the bulb to the optic foramen, was surrounded by a tumor, smooth, firm, symmetrical throughout and dark grey in color. The mass was about 7–8 millimeters in diameter. The entire tumor and with it the optic nerve was excised. Due to extravasation of blood into the orbit, the exophthalmus increased markedly after the operation, until the lids could not be closed completely over the globe, and there was marked chemosis of the ocular and palpebral conjunctiva. A keratitis ensued which was probably neuro-paralytic in character

[1] Read before the Eye Section, New York Academy of Medicine, Jan. 16, 1928.

rather than caused by the lagophthalmus. The eye was kept bandaged, the exophthalmus and chemosis gradually subsided and, January 6, 1928, the child was in good health. A slight corneal opacity still remains, the iris is of a lighter color than its fellow and there appears to have been an organized exudate in the anterior chamber.

The following report of the examination of sections of the tumor was given by Dr. Eggston, pathologist of the Manhattan Eye, Ear & Throat Hospital:—

The specimen consists of two slides, each with two mounted tangential sections of an optic nerve and adjacent tumor tissue. The sections measure 23 x 6 millimeters.

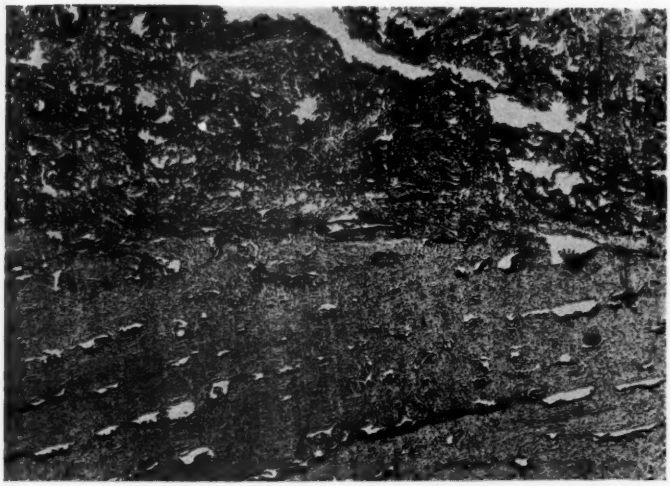
The microscopical examination of the specimen reveals a diffusely enlarged and edematous optic nerve. There are some degenerated nerve fibers, and there is an increased cellularity of the fibers and sheaths. The nerve funiculi are distinctly demarcated with perineural connective tissue which shows many spindle cells. Within the nerve bundles, there are numerous rather large, round, vesicular, nucleated cells. There are numerous dilated lymph capillaries in the nerve. The epineurium is considerably thickened and upon one side it is continuous with the tumor mass.

The tumor mass consists of cellular tissue composed chiefly of spindle and stellate cells. In certain areas the cells are arranged in whorls and, in others, they have an alveolar arrangement. The cells have rather large, vesicular nuclei but no active mitotic cells are found. There are numerous small blood capillaries present in the tumor mass. Large amounts of intercellular fibrillæ are present.

Diagnosis:—Glioma.

Primary tumors of the optic nerve are among the rarest of eye diseases. Thus, H. Sattler found only ten in 400,000 cases and states that Treacher Collins found only two, in 400,000 cases covering a period of fifteen years and Alfred Graefe only three in over 100,000 cases. They were formerly classified under a variety of headings: fibroma, fibrosarcoma, myxosarcoma, etc. Byers, quoted by Parsons, found in 1902 six gliomas in one hundred cases, but the tendency in more recent years has been to regard more and more of these tumors as gliomas. Thus, Hudson in 1912 concluded that 118 out

ILLUSTRATING DR. HERBERT WRIGHT WOOTTON'S ARTICLE, "A CASE OF GLIOMA OF THE OPTIC NERVE."



Section showing relation of tumor (above) to the nerve (below) and the connection of the tumor mass with the epineurium.

Section of tumor showing the spindle and stellate cells with large nuclei arranged in certain places in whorls, in others having an alveolar arrangement.

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of 154 cases gathered from the literature should properly be classified as gliomatous, although only 28 were originally reported as such. Verhoeff is of the opinion that the most common tumor of the optic nerve is glioma and that it is the only primary intra-neural tumor.

Glioma may involve the orbital portion of the optic nerve alone, in which case it is usually benign, and metastases do not occur. Since the optic foramen was not enlarged, this may very well be the status of the case described, and a recurrence is not greatly to be feared. Such a case reported by Hidano was apparently in good health 7 months after extirpation of the tumor. The history of the two cases reported by Weekers in 1926 is similar, one having had no recurrence at the end of five years. However, these tumors may involve the cranial portion of the nerve and chiasm, in which case death has ensued from meningitis following their attempted removal, and cerebral metastases are to be apprehended. This disease is most frequently found in infancy, childhood and adolescence, and it sometimes occurs so early in life that it must be regarded as congenital. Vossius reports a case in which exophthalmus and some strabismus were noticed a few weeks after birth. As exceptions, it may be noted that W. A. Brailey in 1887 reported a "fibroma" of the optic nerve in a man 62 years old. H. Sattler regards this as a case of glioma. The patient ascribed his condition to a blow of the fist received six months before proptosis developed. Two cases are also recorded in which symptoms did not occur until the forty-fifth year.

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SOME OBSERVATIONS OF THE EYE CLINIC OF DR. HENRY T. HOLLAND, AT SHIKARPUR, INDIA.[1]

By Dr. JAMES WILSON CASSELL, New York City.

LAST winter, I had the privilege of visiting the Eye Clinic of Dr. Henry T. Holland. It is said to be the largest operative eye clinic in the world. It is located in India, at Shikarpur, in the Sind. I went as a visitor, but the first day I was there, I was invited to become a member of the staff.

Shikarpur is a place of sixty thousand (60,000) inhabitants, about three hundred and fifty (350) miles north of Karachi, in the Indus River valley. In the summer time, it is one of the hottest places in the world. When I was there during January and February, the days were pleasant and the nights were cool. Occasional sand and dust storms were very disagreeable, the sand penetrating the wards of the compound to their innermost recesses.

Dr. Holland is an English surgeon, with a hospital at Quetta, in Baluchistan,—about two hundred and twenty-five (225) miles from Shikarpur. He comes down each year, during January and February, for six or seven weeks, to conduct this free eye clinic at Shikarpur.

All kinds of eye operations are performed at this clinic, but cataracts outnumber all others.

From January 1st to February 20th, last winter, in about seven weeks, there were twelve hundred and forty (1,240) cataract operations alone.

The first day I was there, ninety-one (91) extractions were performed.

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[1] Presented before Eye Section, New York Academy of Medicine, January 16, 1928.

When we consider,

(1) the conditions under which these operations were performed,
(2) their post-operative care,
(3) their great number,
(4) and the actual results,

we must admit that this is a unique institution.

The HOSPITAL BUILDING is one story in height, and divided into four rooms:

1 for receiving and distributing patients,
1 for dressings, and
2 for operating.

One operating room is devoted to *cataracts* and *iridectomies*. The other, to all other operations.

In each operating room, there are *two* tables, so that *four* surgeons are kept constantly busy operating.

All the rooms are lighted by electricity, but it is not reliable, and we had to depend on *daylight* from a large north window, in operating.

The operating room is a busy place, with a surgeon working at each table, and ten or a dozen patients, squatting down on the floor, their backs against the wall, each waiting his or her turn.

The PATIENTS come from around Shikarpur, and all over the Sind, many of them from long distances;—some come all the way from Karachi, three hundred and fifty miles away. They come on foot, in bullock carts, on camel back, and many come by train.

Their turbans and clothes and bodies may be covered with dirt, as they often are from traveling; but without waiting to give them a bath, or to give their eyes any preliminary local treatment, they are examined. As a rule, when an operation is decided upon, without further ado, they are cocainized, and led into the operating room, and there take their turn upon the operating table. As soon as each operation is completed, and the eyes bandaged, the patient is lifted off the table, placed on a stretcher, and carried across the road, into the compound, and placed in one of the wards.

These WARDS do not resemble the wards of our western hospitals. They are more like a series of sheds or shelters, where patients lie on string beds or cots, placed around the four sides of a court, open in the center, but roofed over for a distance of ten or twelve feet from the wall, with a thatch of palm leaves, reeds and grasses.

Patients in the *first row* are placed on these string beds, which are arranged all around the four sides of the court, but under the shelter of the thatched roof.

Those in the *second row* are placed on the ground, on quilts, at the foot of the beds, but still under the roof.

Those in the *third row* are placed on quilts on the ground, out in the open court, with no protection from above. It rains very seldom at that time of year, and only twice, during my stay, were we obliged to make room under shelter for those placed out in the open court of the compound.

MAKING ROUNDS is an interesting event. We usually had to hurdle or step over those lying on the ground, they were so closely packed together. Generally they were lying covered from head to foot, and it took a person of experience to tell which was the head.

Usually a nurse with a dresser, a sort of orderly, went ahead and uncovered those to be inspected. Another would follow to rebandage and carry out directions.

CASTE.

CASTE. Caste distinctions are so rigid among *Hindus*, that their *food* and *drink* would be *polluted* if anyone outside their own particular caste should even *touch* the *vessel* in which it is contained. Therefore, when a Hindu comes to the hospital he brings along his wife to wait on him and prepare his food and drink. She brings the children, and they bring their pets, which makes quite a retinue with each patient.

The food of the Mohammedan is not polluted in the same way, by being handled, so their rice is cooked in great vats, and stirred by a big scoop shovel, and distributed in large grass mats from which they fill their bowls.

Sectarian prejudice and class jealousy are so strong in

India, at the present time, that Hindus and Mohammedans have to be kept entirely separate, and in different courts.

Operating Room.

In the operating room, each operator has an assistant to retract the lids while the lens is being delivered.

A Registrar records the operation, its form, and anything abnormal.

After *each* operation, *every instrument* is *sterilized*—the cutting instruments in alcohol, all others by boiling.

Just before operating, the *tension* is taken, with a McLean tonometer, in *every* case. Glaucoma is very common. The tonometer findings usually decide the form of operation. If of *normal* or only *slightly increased* tension, an *Intracapsular* operation is done. But if there is *marked increase* in tension a capsulotomy is usually done.

Technique.

Dr. Holland's technique is about as follows:—

After cocainizing,

(1) The *eye* and *surrounding area* are *cleansed* and made as *aseptic* as possible, with Bichloride 1:2000. The *cul de sacs* are *irrigated* with the same solution, special attention being given to the lid margins, the puncta, and the cilia.

(2) The cilia are trimmed off the *outer third* of both lids.

(3) The Tension is taken.

(4) A speculum is inserted and the *section* is made at the limbus, taking in the upper half.

(5) The Iridectomy is performed.

(6) Then, (a) If the *tension* is *high*, a capsulotomy is made with the point of a Graefe knife, and the lens delivered through the capsule incision.

(b) If the *tension* is *normal*, after the iridectomy, the speculum is removed, the Fisher lid hooks inserted under the upper lid and the lens removed in its capsule. He usually does a Smith operation.

(7) Then the pillars of the coloboma are carefully replaced, as well as the corneal flap.

(8) The eye is closed and a wet pad, dipped in Boric Acid Solution, placed over it.

(9) Both eyes are bandaged, and the patient removed to the compound.

Time.

Repeatedly I timed him, and found he averaged about three minutes, from the time he began to irrigate the conjunctival sac, until the operation was completed.

Post Operative.

All uncomplicated intracapsular cases had their first dressing done on the *fifth* day.

Usually on the *seventh* day they were given a shade over their eyes, and

On the *ninth* day they were generally allowed to go home. Their vision was always taken before going.

Final Results.

In the twelve hundred and forty (1,240) cataract operations, performed last winter, there were less than one per cent. (1%) of infections; between two and three per cent. (2%–3%) of vitreous loss; about three per cent. (3%) of prolapse of the iris, or incarceration of the pillars of the coloboma.

Regarding Infections.

I would say that Dr. Holland himself is *strictly aseptic*, both with his hands and instruments. But when we consider the condition of the patients, and the condition of their eyes, when they arrive at the clinic, we should expect a larger percentage of infections.

Vitreous Loss.

He is an *adept* at delivering the lens in its capsule, but displays remarkably good judgment in knowing when to *desist*

from attempting an intracapsular, and resorting to a capsulotomy when he encounters a tough zonule. Very rarely does he lose vitreous in his own personal operations.

Iris Prolapse.

He is particular about his technique, and always carefully replaces the pillars of the coloboma, after delivering the lens. Not a few of his iris prolapses were found in cases where inquisitive patients or their friends had disturbed the dressings.

Choroidal Hemorrhage.

Choroidal hemorrhage occurred in a few cases of high tension, even where capsulotomy had been done.

In conclusion I would once more call attention to the magnitude of the work done at this clinic, to the conditions under which it is carried on, and to its remarkably successful character, all of which, as I have already stated, make it a truly unique institution.

FUNDUS AS A DEFINITE INDEX TO ARTERIAL DISEASE WITH ANALYSIS OF 100 CASES.[1]

By Dr. S. A. AGATSTON, New York City.

IT is universally recognized that marked changes of the retinal arteries are associated with changes in the organs, particularly the kidney. That changes in the fundus can be found in general arteriosclerosis and interstitial nephritis in their incipiency, and that such changes are synchronous with corresponding changes in the kidney and other organs has not been sufficiently emphasized. While much had been written, the trouble has been that the opthalmologist had not delved deeply enough into the medical aspect, and the internists are not sufficiently keen with the ophthalmoscope.

It is my contention that sclerosis of the arterioles of the kidney and brain cannot exist in the absence of similar condition of the retinal arteries and normal retinal vessels definitely exclude interstitial nephritis of the sclerotic type (small contracted kidney). Nephritis, or "nephrosis," such as is encountered following one of the infectious diseases, or is possibly caused by focal infection and is characterized by considerable albuminuria, may exist without arterial disease. The fundus in these cases may be normal or may sometimes show albuminuric neuro-retinitis without any, or slight sclerotic changes in the retinal arteries. To support this theory, I have compiled a number of cardio-vascular cases on the medical service of Montefiore Hospital with study of blood pressure, blood chemistry and kidney function in relation to fundus appearance.

Changes in the retinal arteries correspond to the degree of development of arterial disease in the organs and the brain.

[1] Read before Eye Section, New York Academy of Medicine, January, 1928.

To facilitate their recognition I have numbered them in the order of their usual appearance, as pointed out in a previous paper.

Degree No. 1. Increased light reflex. In young individuals, the light falling upon the arteries and veins creates a white line coursing unsteadily on top of the vessels. This is a manifestation of what is known as juvenile reflex, and is accompanied by a silvery shimmer on the retina. Older patients with normal arteries show only a slight reflex.

No. 2. Reflex more marked. Color and caliber of the arteries unchanged but there is a slight pressure effect on the veins. By the last is meant the effect on the outline of the vein as a sclerotic artery passes over it. In proportion to the degree of sclerosis, the lumen of the vein is attenuated, or the vein may actually disappear for a short distance as it passes under the artery. In this second degree, there is only a slight narrowing of the vein.

No. 3. Copper wire arteries. The arteries become slightly narrower than normal, somewhat uneven in caliber and decidedly paler. Pressure effect on the veins is marked.

No. 4. The same as No. 3 with tortuosities of the small branches of the arteries and veins, particularly in the region between the disk and the macula.

No. 5. The same as No. 3 and No. 4 with marked tortuosities with small hemorrhages and exudates.

No. 6. Perivasculitis, with occlusion of small branches.

No. 7. Edema of the disc with larger exudates and hemorrhages, atrophy of the retina, with low grade pigment proliferation.

Blocking of the central retinal artery is most likely to occur in arteriosclerosis of the second, or third degree.

Very moderate sclerosis of the retinal blood vessels may be accompanied by very advanced sclerosis of the choroidal blood vessels. The vessels of the choroidal mosaic become very pale and sharply defined. Visibility of the choroid is due to atrophy of the pigment epithelium.

Average blood pressure corresponding to degree No. 1 is normal; No. 2, 150 to 160; No. 3, 170 to 180; No. 4, 190 to 200; No. 5, 6, and 7, over 200.

The early changes are frequently unnoticed and fundus examination is called negative. In the absence of changes in the retinal arteries, high blood pressure is most likely functional, and kidney normal.

The first most important definite sign is the pressure effect on veins. This is usually accompanied by pale copper wire arteries. In the presence of some pressure effect, with large arteries of good color, there is no elevation of blood pressure.

In examination for life insurance a careful scrutiny of the fundi would yield the examiner considerably more information than just taking the blood pressure. The latter can be temporarily reduced by a starvation diet; even the urine can be freed of a trace of albumen, but the fundus picture is constant. I would, for instance, consider a man, 50 years of age, with normal retinal arteries, a very good risk in the absence of any surgical pathological condition. On the other hand, a patient showing change No. 2 in his retinal arteries would surely be a poor risk, even though negative otherwise. His arteriosclerosis is apt to progress; he may develop blocking of a cerebral artery or vein, or even blocking of his coronary artery. This inference is drawn from the occurrence of so many cases of hemiplegia and angina without high blood pressure, endocarditis or syphilis. Severe attacks of cerebral apoplexy are more frequent in patients without very high blood pressure, and are usually unexpected. A careful examination of the fundus in these cases would disclose incipient arterial disease.

What may occur in cerebral arteries and veins can be visualized in cases of blocking of the central retinal arteries and veins. A very good demonstration of what may happen was seen in a patient who came to the New York Eye and Ear Infirmary, Dr. Shine's service, with a blocking of the central artery. While taking his blood pressure, which was found to be 160, he developed a hemiplegia. Clinicians will as a rule distinguish between attacks of "stroke" in the presence of low blood pressure and apoplexy in the presence of high blood pressure. The former they ascribe to thrombosis of cerebral artery or its branches, whereas the latter to hemorrhage caused by rupture of a cerebral artery. Patients with high blood pressure are more apt to have hemor-

rhage from the smaller branches of arteries or veins, or else they may have blocking of the smaller branches, as in the presence of high blood pressure, they are not likely to develop a thrombosis in the larger trunks unless the disease is very far advanced, because the high blood pressure creates a strong "vis a vergo."

When we see what happens in the retina as a result of thrombosis of the central vein, *i.e.* very large hemorrhages, in the presence of moderate elevation of blood pressure or low blood pressure, it seems to me that a similar accident may occur in the brain. Of course, free collateral venous circulation of the brain must be taken into consideration. A thrombosis of the central artery results in atrophy of the retina. In the brain, blocking of a cerebral artery results in softening of the brain. The extent of the disease depends on the size of the blood vessel which was blocked. It would seem logical to come to the conclusion that severe attacks of apoplexy with hemiplegia are not always caused by hemorrhage from a ruptured artery, but may be caused either by thrombosis of a large artery or vein, in the absence of very high blood pressure or *in the presence of a rapid drop in blood pressure*, such as might be caused by shock with weakened heart action. In fact, strong doubts may be entertained as to the frequency of rupture of larger branches of arteries. These conclusions are drawn merely from careful study of the behavior of the retinal arteries. I have never seen a rupture of the retinal artery, except at the terminals. Atheroma of the arterial wall, I believe, occurs only in the larger arterial trunks and may exist without sclerosis of the arterioles. Further study of the retina disclosed that cases of peripheral arteriosclerosis such as is found in old people is not necessarily associated with changes in the retina. Patients with this condition may have normal blood pressure and normal kidney function, and longevity is not affected. This observation may again be visualized in the eye, where we find that peripheral arteriosclerosis, while not affecting the retinal blood vessels is, however, often marked by sclerosis of the choroidal blood vessels, with secondary atrophy of the retina and pigment epithelium. The choroid is clearly seen and the choroidal blood vessels are very light red or almost white in color and

very sharply outlined, giving the picture of a marked "tessellated" fundus. (Islands of choroidal pigment are clearly seen.)

There is another type of arteriosclerosis which might be called endarteritis obliterans, which is characterized by extremely narrow arteries sometimes with perivasculitis, with optic and retinal atrophy without the usual picture of tortuosities, exudates and hemorrhages. This type is associated with blood pressure of over 200. Blocking of the central retinal artery or its branches may occur, usually, in the event of temporary dropping of blood pressure.

Such narrow arteries may sometimes be seen in retinitis pigmentosa. In the latter, blood pressure is not elevated.

Very high blood pressures are sometimes seen in patients who show hardly any sclerotic changes in the retinal arteries. In these cases, blood pressure is due to contraction or spasm of the arteries, and is marked by frequent variations in degree of elevation. Urine is usually normal. Contracted arteries with irregular caliber, though good color, may sometimes be seen in the fundus. This is generally known as essential hypertension. Patients may have transient cerebral attacks or intermittent claudication in the presence of very high blood pressures, and sometimes normal blood pressure. Contraction of the arteries, brought about possibly by some change in the blood chemistry (such as excess of cholesterin,) internal secretions, or presence of bacterial toxins in the blood. The mechanism may be analogous to intermittent asthma. I found a number of this type of patients in whom the diagnosis of hypertension with general arteriosclerosis was made. The appearance of the fundus put a different aspect on these cases. That contraction of the arteries can occur is a proven fact. I can cite instances of such occurrence which I was fortunate to observe during the entire process.

Patient, H. N., a young man 23 years of age, came to my office complaining of attacks of blindness in the lower field of his right eye, which during three weeks previous to his visit, recurred at intervals of three or four days. This blindness would last one or two minutes. His fundi were normal, and his retinal arteries were normal; his blood pressure was normal; he had an old mitral lesion. While

I was explaining to the patient that these attacks were caused by a transitory disturbance of the circulation, the patient suddenly called my attention to the fact that he was just having one of these attacks. Taking him into the dark room without delay, I noticed that the upper branch of his retinal artery disappeared from view. While observing the fundus with the ophthalmoscope, I noted a sudden rush of blood into the invisible artery. Simultaneously with the reappearance of the artery, the patient exclaimed that he again could see.

Recently, I saw a case with similar attacks in a young woman, M. B., 23 years, also with an old mitral lesion. For some reason or other, contraction was limited here to a branch of one retinal artery.

A similar contraction of the coronary artery would cause angina. We do see cases of angina pectoris, without elevation of blood pressure, which are relieved by nitroglycerin. It would be easy to understand that such contraction, partially sustained, in many places of the body, particularly in the kidney, and other organs, would produce a functional hypertension. Such contraction, prolonged, could produce symptoms and damage caused by blocking of an artery. This would explain cerebral cases without arteriosclerosis. In actual arteriosclerosis, connective tissue replaces the unstriped muscle of the arterial wall, contraction becomes gradually less possible, and persistent high blood pressure is then brought about by the narrowing of the lumen of the arteries caused by sclerosis, coupled with hypertrophy of the heart.

Finally, I want to state definitely that in post mortem examination of the retinal arteries, the microscope does not disclose moderate changes, although such were recognized with the ophthalmoscope.

In going over 100 cases which the writer compiled and tabulated, we can draw the following conclusions:

1. Blood pressure is generally in proportion to the changes in the retinal arteries.

2. High nitrogen content of the blood is also usually in proportion to changes in the retinal arteries. Very high blood pressures seem to hold the urea nitrogen and kidney function down to normal in some cases.

3. In cases of advanced retinal arteriosclerosis, urine is

never normal; in incipient and moderate cases it is frequently normal.

4. In the presence of normal retinal arteries, high blood pressure is generally functional and is usually associated with normal blood chemistry and kidney function.

5. In cases of auricular fibrillation, blood pressure is frequently higher than one would expect from the appearance of the retinal arteries.

6. Patients with incipient and moderate arteriosclerosis without much elevation of blood pressure may suffer from hemiplegia, or coronary disease with angina, probably caused by *either contraction or spasm of the arteries or thrombosis of a branch of artery or vein.*

7. Retinitis, in the presence of good arteries, is caused by toxemia or change in blood chemistry, such as is found in diabetes. It is not necessarily accompanied by elevation in blood pressure. It has no bearing on prognosis, if the general condition is under control. Thromboses of veins give the fundus the spurious appearance of advanced arteriosclerosis, and careful inspection of the vessels of both eyes will give the examiner a clearer conception of the actual condition of the arteries. The same may be said of fundi showing blocking of the c. ret. art. through spasm or embolism.

8. Incipient changes in the retinal arteries are frequently unrecognized, even by many ophthalmologists and fundus reports are often either misleading, or insufficiently helpful.

9. Contractions of the arteries in normal and incipient cases may be toxic in origin. In these cases clinicians could concentrate on removal of cause, and lowering blood pressure without risk to patient.

In concluding, I wish to stress the importance of a close association of the internist and ophthalmologist, whose careful ophthalmoscopic report, as an aid in diagnosis and prognosis should be taken more seriously than in the past.

I also wish to thank Dr. Florence Frankel, and the interne staff of Montefiore Hospital who were extremely helpful in collecting my statistics.

A list of tabulated cases will be reported at another time.

OCULAR DEPIGMENTATION ACCOMPANYING GENERALIZED VITILIGO.[1]

Report of a Case.

By Dr. DAVID WEXLER, New York.

(With two illustrations on Text-Plate XX.)

VITILIGO (1) is an acquired cutaneous achromia characterized by variously sized and shaped single or multiple patches of milk white color, usually with hyperpigmented borders. The hair may or may not be depigmented. Syphilis, nervous and infectious diseases, and malignancy are given as causes.

Patches of depigmentation of the iris, lids and surrounding skin have been observed for years. Wyss (2) described local vitiligo of the skin in herpes zoster ophthalmicus in 1871. Nettleship (3) observed vitiligo of lids and cilia in sympathetic ophthalmia. Recently Baldino (4) and Löwenstein (5) have noted vitiligo of the skin of the lids and of the iris following herpes iris. Gastgeiger (6) has reported whitening of the cilia (poliosis) in a case of iridocyclitis and interstitial keratitis.

Gilbert (7) observed the first instance of generalized vitiligo with disease of the eye. His case presented severe bilateral iritis, then optic neuritis which subsided, with good vision. The skin showed vitiligenous patches, while all the hair, even the eye lashes, was depigmented. In 1911, Erdman (8) related the case of a dark-haired male, with blue irides, characteristically albinotic fundi and finally chorio-retinitis. Generalized vitiligo of the skin gradually developed. Komoto (9) reported a remarkably similar case in which the eye lashes, and the skin of the hands and feet turned white soon after the development of a "blonde" fundus and diffuse choroiditis.

[1] Case presented before the Section on Ophthalmology at the New York Academy of Medicine, January 16, 1928.

Hanssen (10) noted triangular brown pigment spots in the conjunctiva in generalized vitiligo.

The case to be reported is interesting on account of its long history and unusual findings.

Report of Case.

Mrs. A. W., age 45, colored, born in New York City, was admitted to the service of Dr. J. Wolff at Mount Sinai Hospital, October 28, 1927, complaining of gradual loss of vision, without pain, in the left eye over a period of 10 years. A year ago she began to suffer pain in the right eye, vision being so reduced in this eye that she finally came for treatment. Fifteen years ago, the skin of her hands and feet began to turn white; gradually all skin areas of the body have become involved. She is married, has had five miscarriages; her three children and husband have had neither skin nor eye disturbances.

Examination is negative, save for diseased teeth and the skin and eye conditions to be described. There is no evidence of tuberculosis. Blood and spinal fluid Wasserman are negative. Hgb. 80%, RBC. 4,350,000, WBC. 7,000, Polys. 63%, Lymphs. 35%. Blood chemistry figures including urea N, cholesterol and blood sugar are within normal limits. Urine negative.

Skin of the right upper lid has an irregular patch of depigmentation. There is a narrow zone of brown pigment about the corneal limbus, but none at the site of the perforating conjunctival vessels as is seen in negroes. There are fading gray spots on Descemet's membrane; anterior chamber is very shallow. Iris is bombé, atrophic, covered with a dirty gray membrane. A dense synechia binds the pupillary edge of the iris to the lens nasally. A thin membrane fills the pupil. Some reaction to the light persists. Media and fundus cannot be seen. Tension (Schiotz) 36; vision $\frac{20}{200}$.

The conjunctiva of the left eye presents a bleached appearance; the pigment zone about the limbus is missing in this eye, as is also that at the site of the perforating vessels. A view of the media and fundus is obstructed by a dense pupillary membrane which adheres to the lens all around. With the loupe several gray spots are seen on the iris. Slit-lamp shows these to be irregular areas of atrophy of varying depth, exposing gray unpigmented iris stroma. Iris is fixed to light. Tension 36; vision limited to hand movements.

Transillumination by means of the Sachs transilluminator reveals a striking abnormality in both eyes. On applying

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ILLUSTRATING DR. DAVID WEXLER'S ARTICLE, "OCULAR DEPIGMENTATION ACCOMPANYING GENERALIZED VITILIGO."

Right Eye

Left Eye

Scleral transillumination showing chorio-retinal depigmentation.

UNIV. OF MICH.

the instrument to either the upper or lower lid, the entire globe, except the iris, takes on a vivid pink glow and a bright red reflex occupies the pupil. This is accentuated by applying the tip to successive parts of the sclera itself. The iris is opaque to the light and appears in bold relief to the bright grayish-pink of the sclera. The appearance in the right eye is less marked, the pupillary glow is intense and about the iris the sclera is highly illuminated, but the light color shades off into darker band-shaped segments which transmit light to a considerable degree nevertheless. These effects are brought out more vividly by using the tip (without condenser) of the May Ophthalmoscope, imparting additional redness to the illuminated surface. Transillumination of the scleræ of the patient's daughter shows none of these phenomena.

On October 31st, iridectomy was done in the left eye (by Dr. Goldstein); repeated in the right eye on November 6th. In the coloboma of the left eye, a brilliant red reflex could be seen emanating from the fundus, but no details of structure. The diagnosis was bilateral chronic uveitis and chronic secondary glaucoma; bilateral chorio-retinal depigmentation; partial depigmentation of conjunctiva and iris, left eye.

The hair of the scalp is wiry and dark with a few white strands in front. The skin of the forehead is milky white. That of the face has an irregular mottling on a white background. Except in one or two spots, that of the brow, lids, and nose retains the pigment. The skin of the chest and abdomen presents a clear white base with fairly widely separated varying sized brown spots. There is no hyperpigmentation about their edges. The extremities show a similar pattern, except the pigmented areas are larger and more irregular. The palms and soles are almost uniformly brown. Genital and anal furrows are not depigmented. Pubic and axillary hair have the usual negroid characteristics. A diagnosis of generalized vitiligo was made by Dr. Goldenberg.

Discussion.

The attempt to transilluminate a normal eye results in a faint red glow in the pupil. The pigment epithelium of the retina and perhaps the choroidal pigment, are important factors in the obstruction or absorption of most of the light. The negro eye, being deeply pigmented, exhibits therefore very little or no pupillary reflex. This fact renders transillumination in the negro of little importance as a diagnostic

aid in intraocular growths. It is evident then, that a high degree of depigmentation of the choroid and retina exists in this case more marked in the left than in the right eye.

The history of preceding events in the eyes can only be surmised. If the story be correct, the primary symptoms appeared in the left eye 5 years after the onset of depigmentation of skin and run parallel to the skin change for 10 years. It is natural, as Erdman did, to point to a primary disturbance in pigment formation or regeneration affecting both retina and skin. It is doubtful whether the chronic uveitis, or chorio-retinitis (if such existed) is responsible for retino-choroidal pigment disappearance. More likely the inflammatory process is incidental to disturbed pigment function in the eye. While it is true chronic glaucoma may produce isolated spots of atrophy in iris tissue with pigment loss, this effect is local; a severe loss of pigment in the rest of the globe is hardly a glaucomatous sequel. The loss of pigment in the conjunctiva which is exempt from change in uveitis and glaucoma is further proof of a remote cause of the pigment change.

I wish to acknowledge my indebtedness to Dr. Julius Wolff for permission to report this case.

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A METHOD OF DILATING THE LACRYMAL DUCT BY RAPID DILATATION WITH SEA TANGLE PROBES—PRELIMINARY REPORT.[1]

By Dr. ALBERT L. BROWN, Cincinnati, Ohio.

IN an effort to find a satisfactory method for relieving chronic closure of the lacrymal duct, the passage of probes has always been the backbone of the treatment. There have been several complicated operations to re-establish drainage into the nose which produced varying results in the hands of different surgeons.

In infants, passage of a fine Bowman probe and the syringing out of the passage once or twice seems to be a very satisfactory procedure in the vast majority of cases. This is particularly true if there is no purulent retention in the sac, and consequently little scar tissue formation in the duct. The condition in these cases is usually an up-turned valve which is righted by the passage of a probe.

In adults, of course, the situation is quite different, for there is a formation of scar tissue after a chronic closure and oftttimes a nasal turbinate which is frequently matted down over the nasal opening of the duct. The passage of a probe in such a condition usually aggravates the formation of scar tissue and a false passage is always imminent because strands of scar tissue misdirect the probe. This being the situation, repeated passage of probes simply adds more insult to the already traumatized tissues and definitely defeats the end which we are seeking.

It occurred to me some time ago that if a substance which was stiff enough to be pushed into the tear duct could be found which would also swell in moist tissue, that this substance would pass quickly through the many sizes of probes without

[1] These sea tangle probes are to be obtained from V. Müller & Co., Chicago.

the traumatizing affect of repeated passages. Various substances were tried, such as cat gut, and some other organic substances which swell in moist tissue, but some were too lax to be easily introduced through the entire duct and the others were brittle, and broke off most annoyingly when they were about half way through.

After numerous attempts with sea tangle, it was found that a piece fashioned into a probe about $3\frac{1}{2}''$ long and the caliber of a No. 1 Bowman Probe, could be inserted into the lacrymal passage with very little difficulty. In the average warm moisture of the lacrymal duct this probe was swelled to about six or seven times the original caliber.

The technique of insertion is very simple. A few drops of 4% solution of cocaine are introduced into the lacrymal passage with a lacrymal syringe. After sterilizing the probe by a rapid but thorough washing with alcohol applied to a sponge, white vaseline is rubbed over as a lubricant. The operator then grasps the probe near its tip with a mosquito hemostat and inserts the tip into the lower punctum. By short rapid movements of releasing and grasping the probe, an insertion is effected before swelling has a chance to retard the progress. A strand of thread which has been passed through the upper end through a tiny hole bored near the extremity, is left hanging out to be grasped when removal is desired. The probe may be left in for any length of time, depending on the amount of dilatation desired. I have found that one-half hour to an hour is sufficient. When the probe is to be removed the thread is grasped and pulled.

In the presence of infection of the sac it is well to attempt to quiet the condition first, before using this method of dilatation. The rapid swelling of the probe can apparently aspirate pus into the lower part of the duct and spread infection. This creates a new source for the formation of scar tissue and makes the condition more difficult to deal with. It is best to syringe the lacrymal duct out every day with a solution of $\frac{1}{8000}$ metaphen, giving the patient a solution of the same strength to use at home as an eye wash, until the infection seems to have subsided. I have never found any difficulty if this course were pursued because there is apparently no danger even in the presence of a mucous discharge.

This procedure in twenty cases has shown itself to be a harmless and quite effective method of dilating the tear duct. Whether permanent relief is to be gained in chronic stricture cases, remains to be seen. Before a method of this sort can prove itself years must elapse. The greatest advantage of this method is the necessity for practically only one passage of a probe which of itself is not very traumatizing, for it is a soft, rather flexible substance, and becomes even more softened when it becomes moist and swollen. The swelling, however, is effective enough to definitely dilate the softer tissues of the duct and tear adhesions without causing the production of more scarring. It has long been the custom in many quarters to insert metal probes several times a week, in these chronic cases, trying to introduce a larger probe each time in a desperate effort to permanently establish drainage. Certainly, even if the initial introduction of this substance does not prove effective, the second passage or even the third is not going to produce the irritation that frequent probings by metal instruments would.

This preliminary report is based on the use of sea tangle probes in twenty cases. The ages of the patients range from 18 to 58. The number of passages required and the results finally obtained depended a great deal on the length of time the passage had been occluded, and upon the condition of the patient's nose. Attention in every case was given to the patient's nose. A rhinologist was consulted as to the occlusion of the nasal end of the duct by swollen turbinates, adhesions and the like. After nasal conditions were either declared negative or corrected, the first passage was made, after 4% cocaine instillation in the duct.

The first patient on whom this was tried was a woman 40 years of age, who had had an epiphoria in the right eye 1½ years, and in the left six months. She gave a history of having had probes passed two or three times a week for several months, and she wished something radical done, if necessary, to permanently stop the tearing. I felt that further instrumentation was useless, so I immediately decided to try a sea tangle probe. The probe was inserted in the right tear duct and allowed to remain for 20 minutes, after which time it was removed by traction. The next day the patient returned and

the tear duct was syringed with 4% cocaine solution. The duct seemed patent and the patient was allowed to depart and report again in a week, having observed all symptoms, and especially the possibility of the return of epiphoria. At the end of the week the patient declared that the right eye was free from tearing but that the left eye, which had been untreated, was still occluded. A probe was then inserted in the left duct and allowed to remain ½ hour. The following day there was still epiphoria and another probe was introduced. This was allowed to remain for 30 minutes more and the patient again told to return the following day. This time the epiphoria had quite disappeared. About six weeks later the patient returned complaining of a "watery left eye." This was in the presence of an acute coryza. The nasal edema had apparently obstructed the lower end of the duct. After suitable attention to the nose, drainage was again established.

The foregoing experience encouraged the further trial of the method. The next three patients were young adults. One, a man with chronic atrophic rhinitis, did not respond at all until a scarred, rather shrunken left turbinate was infracted, after which a retention of a probe for one hour alleviated the epiphoria for three weeks. He returned with a recurrence which could not be retarded for longer periods than a month. This, however, was quite a relief because he had had more painful and frequent instrumentation with less relief.

A fair tabulation of the results of this investigation cannot, as yet, be made. Each case has to be based entirely on its individual merits because, while one may be a simple closure, another may be complicated by scar tissue, infection or a chronic nasal condition. The important item in each case is the length of time since the last passage. It will be noted that in the case of repeated passages, the interval lengthens and finally becomes very long. The chart is merely to serve in the stead of unnecessary case details.

In subsequent trials, over an extended period of time, it may be necessary to change the technique, the time element, or even the material used, but I believe that the principle is sound and I offer this as a primary report to be followed later, after the method has had a long enough trial.

Case No.	*No. of Passages*		*Average Time Interval Between Recurrences*		*Time Since First Passage*	*Time Since Last Passage*
	R.	L.	R.	L.		
1	1	3	—	2 days	20 months	19½ months
2	—	2	—	2 weeks	19 months	3 months
3	1	—	—	—	19 months	19 months
4	5	—	1 week to 2 months	—	18 months	12 months
5	—	3		1 month	18 months	15 months
6	10	4	2 weeks	4 months	18 months	R. 8 months L. 10 months
7	4	—	6 weeks Chr. nasal condition	—	17 months	5 months
8	2	—	5 months	—	15 months	10 months
9	1	1	—	—	15 months	15 months
10	1	—	—	—	14 months	14 months
11	8	4	1 day nasal injury	1 day	12 months no relief	10 months intranasal advised
12	5	2	2 weeks	6 months	12 months	6 months
13	2	—	3 months	—	11 months	8 months
14	1	—	—	—	11 months	11 months
15	—	5	—	2 weeks	10 months	7 months
16	2	—	3 months	—	10 months	7 months
17	—	4	—	2 days	No relief to present time. Opened but does not remain so.	
18	4	—	1 week	—	6 months	4 months
19	—	1	—	—	4 months	4 months
20	2	—	1 month	—	4 months	2 months

AIR EMBOLISM OF THE RETINAL VESSELS.[1]

By Dr. HANS BARKAN, San Francisco.

(With one figure in the text.)

THE clinical picture of air embolism is not an unknown one to the general surgeon who has had some experience in chest surgery. Even to him, however, it is not a frequently occurring incident. For the ophthalmologist, although the ocular manifestations are marked, it is a rarity. The tragic picture of air embolism, often terminating fatally, occurs far removed from the ophthalmologist's usual haunts.

Most surgeons are not aware that prompt fundus observations may be of importance to them in these cases; or that any fundus findings of interest occur. There have necessarily been few observations of the fundus in air embolism, as many of the cases end fatally. There are few observations regarding the vision of those that have recovered; scanty mention of the subject in ophthalmological literature; and few experimental observations.

"It would probably not occur to any physician to suggest that air emboli following such common technical procedures as puncture to clear up the nature of a contusion in the thoracic cavity, or irrigation of a long standing empyema cavity with an antiseptic solution, such as bichloride of mercury, hydrogen peroxide, or Dakin's solution, or change of position of the tube draining such a cavity; need cause complications that could endanger the life of the patient." Such is the opening statement of an article by Carl Schlaepfer, entitled:—"Air embolism following various diagnostic or therapeutic procedures in diseases of the pleura and the lung"—in the *Johns Hopkins Hospital Bulletin;* September, 1922.

[1] Read before Am. Ophth. Soc., Quebec, June, 1927.

It would probably also not occur to the ophthalmologist that such procedures could be followed by almost immediate blindness. The history and ocular findings of such a case, with some observations on the appearance of air embolism, as observed experimentally, may therefore be of interest.

Before proceeding with the case report, an abbreviated review of the main clinical signs observed in cases previously quoted in the literature is in order.

In 1864, Roger, a French physician, first called attention to the condition in his observation on a child of eight and a half years of age; suffering from chronic empyema; who became unconscious following an irrigation of the cavity with an antiseptic solution; and in whom blindness occurred. Since that time it has been noticed that patients complained of visual disturbances, headache and dizziness, following chest and lung operations; these initial signs being followed by chronic or tonic contractions of one, or more, limbs. These motor troubles may be accompanied by eye signs:—dilated pupils on the affected side, or on the opposite side, or on both sides; followed by Cheyne-Stokes respiration and accelerated pulse. Death often terminates the picture. At autopsy there are often no positive findings. In a series of cases reported by Schlaepfer, the general picture of which was as just described, eye disturbances are noted as follows:—Spasm with deviation of the eyes upward . . . pupils narrow and not reacting . . . fixed pupils . . . eyes deviating to the right . . . eyes and head turned to the left . . . pupils at first contracted, later dilated . . . dilated pupils . . . eyeballs turned upward and widely dilated pupils . . . patient cries out saying that "he feels giddy or ill; that he has black spots before his eyes; that he is suddenly totally blind."

Of the approximately sixty cases reported in the literature, I will, for the sake of illustration, select only a few which showed marked ocular signs. One of these described by Schlaepfer (a case of blindness for three days following exploratory puncture of the lung) was as follows:—The patient became unconscious the moment the lumen of the exploratory needle in the lung was open to the air. Pupils wide, immobile. This condition lasted a few minutes only. In the course of two and one half hours he recovered slowly. He

stated that he was blind. Because of suspected embolus of the central retinal artery an ophthalmoscopic examination was made next day;—result: normal fundus. For three days following the operation the patient stayed totally blind. On the fourth day vision returned slowly. Dizziness prevented his reading for two weeks. The fundus stayed normal. It was six months after the operation before his vision became normal. No mention is made of fields of vision or examination for scotomata.

The first case in which ophthalmoscopic examination disclosed what we now know to be the typical picture of retinal air embolism is mentioned by Von Weber. The case was one of a man twenty-five years of age, who at twenty-one had diphtheria, followed by pleurisy and lung abscess. At the first operation the right lung was exposed and an attempt made to cause adhesions by tampons. The second interference was with the Paquelin Cautery. Suddenly a fine whistling noise was heard and patient called out:—"I feel sick";—sudden vomiting, unconsciousness, wide pupils and decided nystagmus followed. Slight tonic convulsions of the right arm occurred. Dr. Becker observed air bubbles in the fundus which were seen as "silvery glittering rods." They were seen mostly in the arteries of the retina. Patient died. At autopsy an erosion of a branch of the pulmonary vein was found lying in indurated lung tissue. The arteries of the brain were full of air. In some other autopsied cases, in addition to the air, the picture of a hemorrhagic encephalitis was seen with small areas of cerebral softening. This was the first positively diagnosed case of retinal air embolism.

The first case observed ophthalmologically was probably one of Raynaud's; although a diagnosis of air embolism was not made. One half hour after the patient recovered from unconsciousness, he noticed that he did not seen well. The eyes were fixed; the pupils dilated maximally. The fundus observation, in conjunction with Panas, was as follows:—The papilla of the right eye surrounded by a circle of serous exudation. The veins are broad; in other parts, they seem as if pinched and empty of blood and show no pulsation. The arteries are very narrow; the nasal half of the papilla empty of blood and dull white in color; whereas the temporal half

is of a pale rose with poorly defined edges. Next day the blindness persisted. Another ophthalmoscopic examination showed both papillæ to be a dull white color. The arteries were still very narrow. Curiously enough another washing of the pleura was undertaken, during which the patient again became unconscious; no corneal reflexes; deviation of the eyes to the right. Maximal dilation of the pupils;—coma. This condition remained unaltered for three hours, when a number of characteristic epileptiform attacks took place; during one of which death occurred. A section of the brain was completely negative. The clinical picture is so typical that these descriptions will suffice to point out when air embolism of the retinal arteries may take place and be observed. In the days before air embolism was recognized as a cause of blindness, pleural and vagus reflexes, even hysterical manifestations were thought to be concerned in this condition. The mechanics, however, of both the general collapse with its varied picture, as well as of the blindness, is as follows:—as outlined by Schlaepfer:

"In the wall of an empyema cavity with its underlying, chronically inflamed lung tissue, in the neighborhood of every active infection in the lung, the blood vessels are involved in dense fibrous tissue formation. In an earlier stage, when the infection is still active, the tissue shows a certain amount of infiltration. The vessels are suspended between the fibrous strands. There is a constant pull on these vessel walls when these fibers contract, especially on the veins. When their walls are mechanically injured, the veins cannot contract to the full extent, being hampered by their attachments in this inelastic tissue. The musculature of the vein, being weaker than that of the artery, is insufficient; the opening in the wall of the vein persists; from the hollow of the needle, or from the neighboring cavity (pleural cavity, bronchus) air or gas enters; and we have an air embolism. The result following any injury of the vessel wall shows the inability of these veins to contract to the same extent as they would do in normal tissue.

"The blood in the lung gets its impulse from the right ventricle through the pulmonary artery. The sucking effect of the left atrium and the larger veins of the lungs is not

so efficacious. The combined force of these two factors, together with the act of respiration, makes the current continuous. The sucking influence from the larger veins has a minor effect, in proportion as the vein is situated in the superficial parts of the lung, away from the hilus. A lesion of such a vein by an instrument allows the air, remaining under atmospheric pressure (cavity, bronchus), or gas from a pneumothorax, to enter through the opening. The friction exerted by the air upon the vessel wall causes by reflex action a contraction of the muscles innervated by the sympathetic nerves. This contraction is made ineffective by the attachment of the wall of the vein to the encircling fibrous tissue. At the same time this friction of air and blood along the wall stops the blood proximal to the air. This friction and the internal resistance of the air stops the movement in the blood column proximal to the lesion. No further movement is possible, unless the sucking action from a larger branch of a pulmonary vein comes to their assistance. The air embolism does not become clinically evident. This is the reason why this complication is not reported very often. In the different operations upon the chest wall a lesion of the veins happens quite frequently, but it does not make itself evident. The force has to come from a larger vein under negative pressure in the neighborhood of the wounded vein in order to bring the air and the blood by aspiration into the left atrium. The distance between the point of the mechanical lesion of the vein wall and the junction of this vessel with a larger vein under negative pressure, whence the blood in the lacerated blood vessel, together with the air that has entered, can be aspirated, is the essential point in inaugurating the onset of a clinically manifest air embolism."

Air embolism has been produced a number of times experimentally. Weber injected air into the carotid of dogs, obtaining all the clinical signs described as occurring in the human. In these cases as well as in the monkey, air bubbles were seen in the vessels of the retina by Dr. Becker. Stargardt in the sixth monkey so injected, observed the fundus and gives the following description:—"Almost immediately following injection of air into the carotid, one sees innumerable air bubbles and air columns shoot through the retinal arteries.

The air in the vessels shows a silvery gleam. After one or two seconds the retinal arteries are completely filled with air. At the same time the papilla is pale, practically white. Very soon after this there rapidly follows an appearance which is especially striking:—The entire fundus, especially the retina about the macula contains innumerable very fine glittering lines. These lines cross each other in all possible directions. They seem to hang together like spider nets and form an extraordinarily fine net work of the whole fundus, emphatic through its delicacy and minuteness. The middle of the macular region only—that is, the avascular foveola does not present this picture. This network represents the capillaries of the retina. Two or three minutes after injection, a striking dilation of the veins takes place, and in their middle is seen a bright reflex which must be taken as a sign of the presence of air. As an air column is not seen, I believe that the air in the veins moves forward in the middle of the blood stream in the form of the very finest of droplets. At the time this is observed the fine network described fades away and the air in the retinal arteries disappears. Five minutes after the injection the retinal arteries and veins are normal once more and the papilla again shows its normal color."

Stargardt, in an article entitled *Ueber Luftemboli im Auge*, describes in more detail some parts of this picture. The picture, however, was seen in the indirect image, and therefore only magnified four times. He states that the picture in the direct image, with magnification of approximately sixteen times was not possible, because the animals at the moment of injection exhibited spasmodic movements. The observations took place in monkeys, which give a somewhat more definite picture than do rabbits. In the rabbit, due to the fact that vessels occur only in that part of the retina containing medulated fibers, one does not observe the air in the capillaries. In the rabbit, however, especially in the albinotic ones, the changes in the choroidal circulation are seen much better than in the monkey, due to the lack of pigmentation. The changes that take place I shall describe from my own observations, as follows:

In all these experiments I am greatly indebted to Dr. Leo

Eloesser, Professor of Surgery at Stanford University, who performed the experimental surgery, and who first drew my attention to the whole subject.

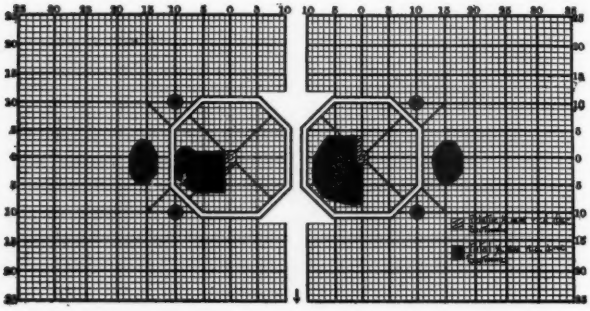
Our rabbits were injected into the right ventricle, it not being possible in so small an animal to make the injection into the pulmonary artery. The air injected into the right ventricle naturally immediately found its way into the pulmonary artery and thence into the lung. The rabbit presented a clinical picture similar to that seen in man. He was seized with clonic convulsions and with direct ophthalmoscopy one sees the remarkable picture of several "rod-like" silvery metallic particles shooting along the retinal arteries. At first a few appear, with an immediate narrowing of the lumen of the entire vessel. The blood in the artery distal to the embolus stops moving; then the blood proximal to the embolus shoves the embolus along; then more appear and are rapidly pushed on so it would seem, by an almost volcanic eruption of air, which within a few seconds delineates the entire arterial tree as a brilliantly discernible shining object. At the same moment one sees numerous air emboli shoot into the choroidal vessels. These become almost entirely white with only a very weak yellowish mixture of color. The return to a normal condition was unfortunately not noted as the rabbits died within a few minutes after the air injection. Stargardt, who injected his rabbits through the carotid observed, however, that the blood column in the arteries moved forward very slowly; in one minute about a papilla diameter, which would correspond to about one millimeter, and that there was marked spasmodic contraction of the arteries. It is certain then, both from the experimental picture as described by Stargardt and as seen and confirmed by myself, and from cases seen by Weber in the human, that an absolutely typical picture of air embolism of the retina exists.

The effects of air emboli have been observed in other fields of surgery; in puncture of the maxillary antrum; in intracranial surgery and neck surgery. Hajek, in 1907, at the German Laryngological Society first pointed out a series of results which might be attributed to such a harmless diagnostic procedure. The air must enter a vein near the puncture hole. It must be assumed that the air bubbles

pass the lesser circulation and enter the arterial system where they, as emboli cause lesions according to the fanciful or accidental paths followed. In one of these cases the examining oculist found the retinal veins normally filled one and one half minutes later; the arteries visible as glistening bands, free of blood. Hajek recalls that more than twenty years ago one of his patients was temporarily blind in both eyes after an inflation of air into the antrum; and Murphy, in 1898, pointed out that in separating adhesions of a diseased apex on the mediastinal side the azygos vein must be given great consideration as it is easily torn and has a double danger:—hemorrhage and air embolus.

My personal experience and ophthalmological study is confined to one case, which I present:—The patient, a male, thirty-five years of age, thinks that he aspirated a bone while drinking soup. A few months later he had violent spasms of coughing with expectoration of exceedingly foul sputum. He now brings up occasional small amounts of blood, constant foul sputum, and coughs continually. Doctor Eloesser considered the possibility of an abscess around a foreign body in the right lower lobe and advised operative intervention. Under good rectal anesthesia four ribs were resected. In a week's time, the second opertion was performed, the lung being sewed to the much thickened pleura and the pleura opened with galvano cautery and then a knife. In the course of the operation the man collapsed alarmingly. Upon leaving the operating table he complained that he could not see; and he was amaurotic for twenty-four hours. Subsequently a bone ¾ by ½*cm* was removed from the right major bronchus. At a third operation further lung abscesses were opened. There was considerable bleeding from a large vessel on the side of a bronchus. Patient collapses, and says:—"I am dying." The violent spasms of coughing suddenly cease. Pupils dilate. He is completely amaurotic. Talks volubly but rationally. Pupils do not react to light. He stays amaurotic for six days; then begins to recognize larger objects. Following this, upon packing with iodoform gauze he is again completely amaurotic for twelve hours. On none of these occasions was I able to see his fundus, as I was not in the city, but I examined the man, who made a

good recovery, some months afterwards. He at that time complained that vision was not as clear as before, and that there was a slight flickering of objects. The fundus examination was entirely negative. The vision was $\frac{6}{10}$ right and left and small print read with difficulty. There was nothing remarkable about his refractive condition. The fields, how-



ever, showed on the Lloyd Stereo-Campimeter the defect as demonstrated. From his history of collapse and amaurosis following an interference with the lung, which we know by analogous cases to be the cause of air embolism, there is no doubt but that air embolism took place, and that the visual defects here demonstrated are the late sequence of cerebral air embolism. That degenerative changes can take place in the brain following generalized air embolism has been shown by Spielmeyer in autopsies on dogs and monkeys operated on by Wever. Spielmeyer states "the first changes take place about fifteen hours after the air embolism and consist of a fine necrosis of the cortical cells and a deformation of the fine nerve endings of the ganglion cells. At this stage a glia reaction is not present. On the third or fifth day following, however, the proliferation of glia cells in these areas is quite an extraordinary one. One sees here also that the changes are in small foci so that in the cortex there are found small areas of devitalized nerve cells; in which areas neurogliar cells are proliferating. The changes are always restricted to small multiple areas. There is never liquefaction of tissue, hemor-

rhage, or necrotic softening. Twelve days afterwards one sees very large glia cells in the place of the cortical cells. The pathology then consists of small cortical areas of partial destruction of nerve elements with substitution by glia cells and no gross areas of necrosis. I believe that the fields presented represent the projection outward of such an area, probably situated about the superior lip of the calcarine fissure and due to air embolism. It is to my knowledge the first lesion of this character described as a consequence of air embolism.

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A REVIEW OF THE LITERATURE ON THE TREATMENT OF SYPHILITIC EYE DISEASE.

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THE purpose of this review is to present the trend of ophthalmologic practice in the treatment of syphilis since the advent of arsphenamin.

The early literature gives evidence of the ill effects of the use of arsphenamin from which not a few ophthalmological impressions have been derived. As early as 1910 Ehrlich's Salvarsan was attracting the interest of ophthalmologists. Their experiences with atoxyl and arsacetin, however, warranted a cautious approach to this new drug and Henderson, (1) in expressing a desire to prevent the general distribution of the drug until its effect upon the eye could be determined, typified a general feeling prevalent at that time. Ehrlich, (2) at this time, however, reported 30,000 cases of syphilis treated with arsphenamin and found only one which showed signs of beginning optic atrophy following the injection. Experiences of many authors also writing at this time or shortly after were in accord with the conclusions of Ehrlich, among them being Dolganoff (3) and Green (4). Such encouraging reports no doubt stimulated a more active interest for the use of arsphenamin by ophthalmologists became increasingly prevalent. It was during this period of uncertainty that McAdams (5) reported questionable results obtained in the treatment of 7 cases of ocular syphilis by the administration of a single dose of arsphenamin. At times as many as 2 injections were given at monthly intervals. The lesions were aggravated following the injection, evidently a Herxheimer effect. Cameron (6) reported the production of a large scar at the site of a primary lesion of

the eyeball following treatment with arsphenamin. A knowledge of the therapeutic paradox no doubt would have prevented this accident. McKee and Courtenay (7) reported very discouraging results in the use of arsphenamin, although only 4 cases were cited in which the ocular lesions failed to involute upon the administration of a total of only three to five injections. Howley (8) also administered but two to three injections of neoarsphenamin as sufficient treatment, yet his results were uniformly gratifying. Uhle and McKinney (9) reported a series of 50 cases of ocular syphilis treated with arsphenamin. In most cases only 1 to 5 injections were given and always at monthly intervals. Progressively from year to year therapists felt more assurance and less fear in the administration of arsphenamin, increasing the dosage, lengthening the courses and cutting down the intervals until methods approximating our present interpretation of intensive treatment were established. As representing this later appreciation of more intensive and prolonged though still incomplete arsphenamin treatment, I cite Hern (10) who in 1921 found that 8 to 10 injections of neoarsphenamin in chronic cases and 7 to 8 in acute cases were sufficient to cure the eye lesions and to render the blood Wassermann negative. In the latter event treatment could be discontinued. Further emphasizing the necessity for prolonged intensive treatment, Stokes (11) in 1926 advocated arsphenamin, mercury or bismuth for one year following the last positive blood Wassermann in early uncomplicated cases of syphilis.

Reports appearing in the literature from 1918 to 1927 have been grouped under subheads dealing with various phases of the treatment of ocular syphilis and an attempt has been made to show the superiority of one method over another. To avoid repetition only a few impartial examples of each phase have been included unless their individual value merited mention.

Ocular Syphilis as a Manifestation of a Systemic Disease.

It is evident from reports that writers in many instances have concerned themselves with the treatment of the ocular

lesions alone. The early writers who have been mentioned gave only a few injections of arsphenamin or neoarsphenamin as sufficient treatment providing the eye lesions involuted. The greater proportion, however, have viewed ocular syphilis as a manifestation of a systemic disease and have treated it as such. Shastid (12) emphasized the importance of the investigation of syphilis as a systemic disease in order to appreciate its relation to the eye. Bell (13) cited the frequency with which ophthalmologists fail in their treatment of ocular syphilis as a systemic disease rather than a localized lesion of the eye. Rea (14) noted that the clinical manifestations in general and not the blood Wassermann were the measure of thorough treatment. Hern, however, stated that a negative blood Wassermann was the sign of sufficient treatment. The treatment of ocular syphilis is systemic according to Douglas. (15) Schussler (16) and Zimmermann (17) also emphasize the importance of this phase of the treatment of ocular syphilis.

Hygiene, Foci of Infection, etc., in the Treatment of Ocular Syphilis.

Little need be said as to the importance of hygiene, foci of infection and intercurrent infectious diseases in relation to the effective treatment of ocular syphilis for mention of this phase is met with in a great majority of the reports of which Hern, Schussler, Bell, Ramsey (18) and numerous others are examples.

The Herxheimer Reaction or Therapeutic Shock.

An example of therapeutic shock was first visualized by McAdams in 1914 following the injection of a single dose of arsphenamin when the lesion which he was treating became aggravated. It is only during recent years that the cause of the Herxheimer reaction has been studied and methods advanced for its prevention. Douglas, for example, advised the use of small doses of arsphenamin in starting treatment if Herxheimer reactions locally in the eye with consequent damage were to be prevented. Hern on the other hand still feels that immediate injection of novarsenobillion will not aggravate the existing local trouble. Finoff (19) reported a

case of bilateral neuroretinitis appearing in a patient following treatment with arsphenamin. Therapeutic shock was considered responsible for the complication, and further administration of arsphenamin in small doses was advised. Benedict and O'Leary (20) detected fresh optic nerve inflammation arising after arsphenamin treatment and considered it a provocative syphilitic process caused by the administration of insufficient dosage. The condition subsided following the use of more arsphenamin. Green (21) stated that ocular lesions appearing after arsphenamin administration should be regarded as a further manifestation of syphilis and treated by more arsphenamin. Stokes advised the use of bismuth or mercury as a preparation preceding arsphenamin administration if Herxheimer reactions with possible permanent damage were to be avoided.

Relapse.

Observations by Zimmerman of 190 cases of iridocyclitis treated with arsphenamin suggested the probability that insufficient treatment was responsible for a large percentage of relapse. Out of the group observed there were 21 recurrences all of which had been insufficiently treated. Carvill and Derby (22) arrived at a similar conclusion. 10 cases of their series of interstitial keratitis insufficiently treated all relapsed. A striking difference in the percentage of relapse was noted by these same authors in a series of 100 cases of interstitial keratitis treated with arsphenamin and a similar number untreated. 3.6 percent of the former group relapsed as against 27 percent of the latter group. Recurrences of interstitial keratitis took place frequently in a patient under local treatment but were improved by the use of mercury according to Cabannes and Montoux. (23) Young (24) reported 5 cases of relapse which resulted from the withdrawal of arsphenamin in patients receiving combined arsphenamin and mercury treatment.

Arsphenamin *vs.* Mercury in the Treatment of Ocular Syphilis.

The treatment of ocular syphilis with mercury and the iodides alone is rarely recorded in the literature and repre-

sents about 2 percent of the material investigated. Hogan and Robertson (25) from their experience with flumerin concluded that mercury was far more valuable in the treatment of the eye complications of syphilis than arsphenamin or neoarsphenamin. Their conclusions, however, were based upon the treatment of only three cases. Harkness, (26) from questionnaires sent to 90 physicians inquiring as to their methods of treating ocular syphilis found that 64 percent used arsphenamin or neoarsphenamin in conjunction with mercury, and 35 percent depended upon mercury alone. This was of course an uncertain method and as the author states was not an index to therapy. Of the entire literature investigated approximately 90 percent of the writers remarked upon and demonstrated the superior value of combined arsphenamin, mercury and iodide medication in the treatment of ocular syphilis. About 50 percent of these reports deal with the treatment of acute inflammatory lesions of the eye. A few examples will be found in the reports of Garraghan, (27) Wurdeman, (28) Lloyd, (29) Lundwic, (30) Miller, (31) Siefelder, (32) Bulson, (33) Green, Early, (34) Young and others. Young stated that arsphenamin was the best drug in the treatment of ocular syphilis and that mercury alone had but little effect aside from its resistance building qualities. Amat, (35) in demonstrating the superiority of arsphenamin over mercury recorded the prompt retrogression of a gumma of the inner canthus of the eye following arsphenamin whereas it had proven resistant to treatment with mercury. According to Rea the best results from the standpoint of the eye, *i.e.*, partial and permanent damage prevented and vision improved were produced by the intensive use of arsphenamin along with mercury and the iodides.

Non-specific Effects of Arsphenamin.

Only occasional mention was found dealing with the non-specific effects of arsphenamin and neoarsphenamin in relation to the treatment of ocular syphilis, although it was evident in some cases of questionable diagnosis or therapeutic test that such an error might have occurred and distorted the author's interpretation of his results and con-

clusions. Stokes outlined in detail this procedure and advised against the use of arsphenamin in the performance of such a test when the possibility of a tuberculous, mycotic or low grade infectious process existed as an etiologic factor. Such a drug as bismuth or mercury was suggested as a reliable substitute for such diagnostic measures. Kolmer (36) likewise noted the non-specific effects of arsphenamin and neoarsphenamin. Benedict and O'Leary reported marked improvement in a series of both non-specific and specific uveitis and retinitis following the administration of arsphenamin. Pfingst (37) demonstrated the non-specific effect of arsphenamin in the treatment of specific and non-specific iritis and cited relapse as occurring more frequently in the latter.

Bismuth.

The use of bismuth salts in the treatment of ocular syphilis is comparatively recent, and its position in the syphilitic armamentaria cannot be ascertained until further and more complete studies have been carried out. However, Cassati (38) reported 5 cases of acute syphilitic eye lesions which responded favorably to treatment with bismuth. Courfein (39) and Babonneix (40) from short series of cases treated had similar encouraging results. These results show the drug to be effective but not distinctive.

Non-Specific Therapy in the Treatment of Ocular Syphilis.

Not unlike that of bismuth the field of non-specific therapy is a new one and the lack of sufficient reports from its use prevent a proper evaluation at this time. Possek (41) treated 84 cases of syphilitic eye disease with non-specific methods. He used typhoid vaccine killed with phenol and made up with approximately 500 million organisms to the cubic centimeter. Of this he injected from .6 to 1*cc* subcutaneously the first day and 1*cc* the second day. 52 of his cases were of long standing and 32 were recent. They included acute eye lesions, interstitial keratitis and optic

neuritis. All cases were benefited. Out of 100 cases of eye disease treated with non-specific methods Uddgren (42) reported the results of 12 cases of undoubted syphilitic origin as inconclusive. 7 cases of scleritis and sclero keratitis in which specific treatment had not been used obtained only temporarily beneficial results. Combined with specific treatment, arsphenamin, mercury and the iodides, the non-specific methods showed a greater percentage of improvement. He used sterile boiled milk. Musy (43) reported excellent results in cases of iridocyclitis and iritis of syphilitic origin with the use of pasteurized milk combined with routine specific treatment. The milk was boiled for 15 minutes and 5*cc* injected intramuscularly every 2 to 4 days. Weichardt (44) reported favorable results with the use of milk in the treatment of acute syphilitic eye lesions. Other reports were by Boas, Lattrup and Anderson, and Freidlander. (45)

Tryparsamide.

Visual disturbances both subjective and objective result from the use of tryparsamide but in such a small percentage as to be regarded as almost negligible. Care, however, is advised in its early administration and the development of objective disturbances is a contraindication to further medication. Tryparsamide in the treatment of neurospyhilis produced 10.2 percent of subjective symptoms and 5.5 percent objective according to Woods and Moore (46). Only 33 percent of a series of 12 cases with already existing syphilitic disease of the optic nerve developed visual disturbance while under treatment with tryparsamide. This altered the earlier views of these authors that syphilitic involvement of the optic nerve was an absolute contraindication to the use of the drug. Lillie (47) reported subjective visual disturbances occurring in 7.8 percent and objective in 3.5 percent. Smith (48) reported 10 percent, of visual disturbances, Ebaugh and Dickson (49) 2 percent, Lorenze, Loevenhart, Reitz and Eck (50) 7 percent, and Crawford 13.3 percent. Stokes and Wilhelm (51) found 2.6 percent subjective and 5.4 percent objective, 91 percent of the subjective disturbances occurring within the first 5 injections. They found that if reactions

did not develop during this time treatment could be carried on without danger as they had given as many as 3 courses of the drug without effect. Cady and Alvis (52) studied 180 patients of which 153 had normal eyes and 27 had optic atrophy. Of the normal group 5.2 percent showed visual disturbances and 1.3 percent actual damage which failed to involute following the withdrawal of the drug as did the former. Tryparsamide injected into rabbits to demonstrate its effect upon the eyes produced no ocular lesions according to Young and Loevenhart. (53) Schwab and Cady (54) in a study of 97 patients on tryparsamide therapy recorded 30 percent subjective visual disturbances. None of these however suggested permanent damage.

Does Arsphenamin as Used in the Treatment for Syphilis Produce Ocular Disturbances?

The consensus is that it does not, this fact being adequately demonstrated by reports, such as that of Young and Loevenhart, who in a careful experimental study of the effects of certain organic arsenicals on the optic tract concluded that organic arsenicals with the amino group or substituted amino group in the ortho or meta position to the arsenic (arsphenamin and neoarsphenamin) did not produce optic lesions in the rabbit. Experimental work was carried out by giving arsphenamin injections to rabbits with a dosage of 25 percent of the minimum lethal dose. Ophthalmological examinations were made before administration of any drug and at weekly intervals thereafter. The eyes were removed for section and studied with negative findings. Wurdeman also stated that he had observed no cases, on the basis of a series of 460 patients under treatment, in which optic atrophy had resulted from intensive arsphenamin treatment. His experimental work with rabbits in observing the effects of arsphenamin on the optic tract confirmed the findings of Young and Loevenhart. No ocular lesions developed. Some of the rabbits were under observation as long as 4 months. Green was of the opinion that arsphenamin did not cause optic atrophy through a direct toxic effect. Ehrlich was able to record only one case of optic nerve atrophy following the use

of arsphenamin. His series under observation totaled 30,000. Dolganoff concluded that arsphenamin did not produce deleterious or hastening effects on optic nerve degeneration or blindness. Gibbard (55) observed but 2 cases of cerebral nerve disease in 1200 patients in which arsphenamin had been administered and that an increase of the dosage caused a disappearance of the trouble. Baruch (56) from a study of 10,000 cases likewise concluded that *choked* disc or neuritis optica in the secondary stage of syphilis had not increased through the use of arsphenamin. According to the author a percentage of 3.67 came within the boundaries as they were given before the arsphenamin era. He cited Wilder and Stehaeline who in 1897 reported 8.5 percent and Fehr in 1912 who gave 2.4 percent. Fordyce and Rosen (57) found that arsphenamin did not adversely affect the optic nerve. Von Hipple (58) likewise was of the opinion that arsphenamin did not injure the optic nerve or retina.

Acute Syphilitic Eye Disease.

Here again arsphenamin or neoarsphenamin is practically universally advocated for the treatment of acute inflammatory lesions of the eye due to syphilis. The articles by Knapp, (59) Curran, (60) McAdams, Cooper, (61) Bell, Ramsey, Carmichael (62) and numerous others are cited as examples.

Interstitial Keratitis.

Conclusive reports as to the most effective treatment of interstitial keratitis are rather infrequent, therefore a consensus is not readily obtainable. Since the report by Carvill and Derby is the most complete and accurate account of the effect of such treatment, it is here considered in detail. Coöperation between the ophthalmological department and the syphilological clinic made possible the study of 200 patients suffering with interstitial keratitis. 100 cases comprised an intensely treated group (a) and 100 represent an untreated group (b). Treatment with arsphenamin, mercury and potassium iodide was given group (a). No treatment

was given group (b). The patients in group (a) were observed for nearly 3 years and those in group (b) 10 years. In all cases the vision was taken for the correction of ametropia. Vision was better or $\frac{1}{10}$ in 50 percent of the treated cases and only 28 percent of the untreated, whereas, vision of $\frac{1}{10}$ or worse was found in 42 percent of the untreated group and only 24 percent in those treated. In group (a) only 15 out of 179 had a vision of less than $\frac{20}{100}$ 3.6 percent of group (a) relapsed as against 27 percent of group (b) 10 cases of the series inadequately treated relapsed. Intensive treatment in the early stage of involvement of the primary eye had a definite influence in preventing involvement of the second eye. Rea obtained favorable results by intensive treatment with arsphenamin along with mercury and the iodides. Kirkpatrick, (63) also had favorable results using combined arsphenamin, mercury and iodide medication. He felt that arsphenamin alone was of very little value. Howley reported marked improvement in 2 cases treated with neoarsphenamin and mercury. Lamb obtained fine results by the use of subconjunctival injections of salvarsanized serum. Green condemned arsphenamin in the treatment of interstitial keratitis due to the frequent appearance of the keratitis in the other eye while under treatment. This observation conflicts somewhat with the above summarized article of Carvill and Derby. Pfingst advised the use of arsphenamin, mercury and the iodides because of the fact that such treatment caused a shortening of the inflammatory period and rapid clearing of the cornea. Bussy and Japiot (64) applied very small doses of X-rays to the eyes in conjunction with the ordinary methods of specific treatment and reported resultant improvement.

Optic Atrophy.

Intraspinal medication appears to offer the most favorable results in the treatment of optic atrophy due to syphilis. Fordyce, (65) for example, gave intraspinal treatment to 33 cases of optic atrophy. No results were obtained in 8 of the cases with almost complete atrophy. Out of 7 cases with

complete atrophy in one eye and partial in the other, 5 became stationary while the other 2 progressed. Out of 17 cases with incomplete atrophy in both eyes improvement took place and the process was arrested in 12 cases. In 5, a further reduction of the visual fields was noted before arrest of the process. In 5 patients arrest of the atrophy had been confirmed for 2 to 5 years. Intensive treatment arrested optic atrophy in a single case reported by Fordyce and Rosen. The treatment comprised 53 injections of arsphenamin, 20 intraspinal injections and 118 intramuscular injections of mercury besides mercury by inunction and potassium iodide. Gifford (66) reported the arrest of optic atrophy in a single case following intraspinal treatment. Gennerich (67) also reported 2 cases of optic atrophy in which the vision ($\frac{6}{24}$ and $\frac{6}{12}$ respectively) was held the same for 5 years after intraspinal therapy. Lamb reported improvement in 2 cases in which he had used subconjunctival injections of salvarsanized serum in conjunction with intraspinal therapy Knapp treated 5 cases of optic atrophy by the intraspinal method and noted improvement in one case. Intracranial injections of mercury bichloride has received some popularity and Gifford, its originator, reported a series of cases treated by this method with actual improvement in several of the cases. A dosage of $\frac{1}{60}$ of a grain of mercury bichloride was given at intervals of 10 days to three weeks making a total of 3 to 4 injections combined with general treatment, arsphenamin, mercury and iodide. Hume, (68) and Viner and McMurtry (69) had similar encouraging results using this method. Moderate success was reported by Iwaschenkoff and Nonne (70) with the intensive use of arsphenamin. Baruch also concluded that arsphenamin was efficacious in the treatment of optic atrophy. Tryparsamide was used by Schwab and Cady in the treatment of 13 cases of optic atrophy. 4 of their series progressed and 9 remained stationary while subjective improvement was noted in some cases. Cady and Alvis using tryparsamide in the treatment of 27 cases reported an improvement in 14.8 percent. 48 percent remained the same while 37 percent became worse. Fever therapy so far has offered no favorable results in primary optic atrophy.

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MYXOMA OF THE ORBIT, WITH CASE REPORT AND ANATOMICAL FINDINGS.

By Dr. H. D. LAMB, St. Louis, Mo.

(With three illustrations on Text-Plate XXI.)

A MYXOMA is a growth composed of mucous tissue. That a true, primary myxoma can occur is attested by several eminent authorities in pathology. The latter group include Ribbert (*Frankfurter Zeitschrift für Pathologie*, 1910, 4, p. 30), MacCallum (*Pathology*, 1920, p. 981), Ewing (*Neoplastic Diseases*, 1922, p. 170), Aschoff (*Pathologische Anatomie*, 1923, p. 681), and Delafield and Prudden (*Textbook of Pathology*, 1923, p. 399).

Mucous tissue closely resembles embryonic connective tissue. The latter consists of round or stellate cells joined by processes, between which there is a large quantity of mucus (mucin) holding interstitial substance. According to Szymonowicz (*Textbook of Histology and Microscopic Anatomy of the Human Body*, 1902, p. 53), this intercellular substance in young embryos is homogeneous, while in older embryos, connective tissue fibrils begin to be formed. This gelatinous tissue is found in the umbilical cord and also in the embryonic cutis.

Ribbert, Ewing and Delafield-Prudden think that primary myxomata must arise from embryonal connective tissue. Ribbert however states that the mucous tissue composing the myxoma differs in many peculiarities from the embryonal tissue; it is more mucoid, richer in mucin and poorer in cells.

Anatomical Forms.—The myxomata are lobulated, nodular, papillary, polypoid or fungoid. They are soft, grayish-white or grayish-red; on section they are found to be smooth,

translucent and gelatinous, containing much fluid and many blood-vessels. The loculation is determined by the growth about blood-vessels, which v. Rindfleisch (*Textbook of Pathological Histology*, 1872, p. 154) found on injection to be of large caliber with few capillaries. The myxomata are frequently very large, sometimes multiple and often contain cysts and hemorrhages. Their encapsulation is seldom complete, the mucinous material tending to infiltrate surrounding tissue. On this account complete extirpation is sometimes difficult and recurrence follows.

Microscopic Structure.—The intercellular substance or matrix of the myxomata varies greatly in bulk and structure. It may be very abundant widely separating the cells. The matrix may be entirely homogeneous, faintly fibrillated or finely granular. The essential cells, although round in particular sections are typically star or spindle shape with processes anastomosing or disappearing in the matrix. The cytoplasm of the cells usually contains fatty and watery droplets.

The Locations of myxomata are chiefly the subcutaneous and intermuscular tissues where the structure is loose and much fat is present. The regions most affected are the thigh, neck, cheek, leg and peritoneum; less frequently the bones, meninges, nerve-trunk, mucous and serous membranes and hilus of the kidney.

The Clinical Course of myxoma is usually of a slowly growing tumor which produces no symptoms except local swelling and pressure. The pure myxomata are in general benign; yet they sometimes grow rapidly and occasionally though rarely form metastases. A myxoma is a rare tumor.

Ocular Forms.—The optic nerve is especially susceptible to myxoma, which here tends to pursue a characteristic course. The tumor occurs chiefly in young subjects. It produces a fusiform swelling of the whole nerve-trunk. Blindness results but the eyeball is usually not involved and extirpation of the tumor is usually successful.

Myxoma of the Orbit is apparently very rare. Only one such case could be found in the literature. E. Fuchs (*Klin. Monatsblätt f. Augenheilk.*, 53, 1914, p. 244) briefly reported a myxoma orbitæ in a 40 years old woman who for 7 years suffered a

ILLUSTRATING DR. H. D. LAMB'S ARTICLE ON "MYXOMA OF THE ORBIT, WITH CASE REPORT AND ANATOMICAL FINDINGS."

FIG. 1.—Appearance of eye before operation.

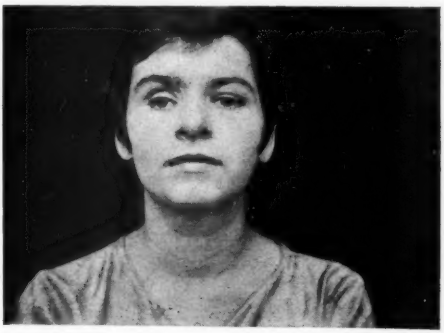

FIG. 2.—Appearance of eye after operation.

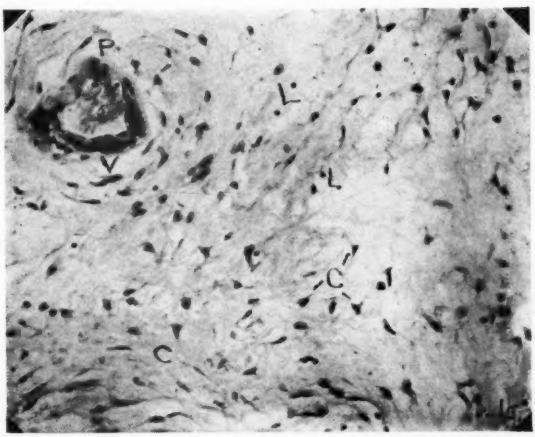

FIG. 3.—Typical section through myxoma of orbit, showing: C—nuclei of essential cells; L—small lymphocytes; V—small vein, containing P—polymorphonuclear leucocytes.

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gradually increasing exophthalmus up to 20*mm.* finally. After the removal of the displaced lacrimal gland and after bisecting the external rectus muscle, the encapsulated tumor of one and one-half times the size of the eyeball was easily shelled out. Vision remained normal. The round growth was enclosed by a free capsule and was a true myxoma. Fuchs added that myxomatous tumors arose from the meninges of the optic nerve or from the lacrimal gland. In his own case, the point of origin was not determined. Klionsky (*Lancet*, 1925, v. 1, p. 126) reported a myxoma extending into the orbit from the frontal sinus. In a woman of 60 years, diploplia had been present for three months and the eye was pushed downward and outward. Enucleation of the eye and canthotomy were necessary before the tumor could be reached. The roof of the orbit was defective and a jelly-like mass protruded from the frontal sinus into the orbit.

Case Report—Clinical and Operative Findings.

By Drs. W. T. COUGHLIN and JOHN GREEN.

M. S. a young girl of 16 years, had noted a gradually increasing protrusion of the right eye for 3 years. There had been occasional attacks of pain of brief duration in the eye; double vision when looking downward and to the left had been present at times. There was no history of injury.

Examination at St. Mary's Hospital revealed a marked exophthalmus (1*cm*) of the right eye, with some displacement downward and outward. No growth could be detected in the orbit by palpation within the bony margin. The motility of the eye was limited in all directions, more markedly inward. The right pupil was moderately dilated but reacted to light. Vision in the right eye was ability to count fingers at 12 feet. Ophthalmoscopic examination showed a swelling of the right papilla to 6 dioptres with veiling of the disc margins; the retinal veins were tortuous but not overfilled. X-ray examination showed an enlargement of the right orbit with obliteration of the right ethmoid cells from pressure, opacity over the right antrum and haziness over the right frontal sinus.

Operation (Dr. Coughlin). A curved incision along the inner two-thirds of the right eyebrow was extended downward and inward for a short distance along the side of the nose. The periosteum of the roof of the orbit was exposed

by blunt dissection but was not incised. The growth did not come from the bony wall, eyeball, optic nerve nor sheaths of the optic nerve. It seemed to spring from the sphenoidal fissure lying nasally and above the optic nerve. It was a grayish, semisolid, glairy and mucoid growth. It filled the orbit, was about 1½ inches in vertical diameter and had to be removed piecemeal.

After-care and Final Results.—Streptococcus infection of the wound followed, controlled by free drainage and an intravenous injection of streptococcus serum and an intramuscular injection of scarlet fever serum. These injections had an immediate beneficial effect upon the infection.

The patient left the hospital one month after the operation. A ptosis of the right upper eyelid, present since the operation was diminishing and movement of the eye inward was improving. Vision was $\frac{6}{10}$. Disc margins were slightly blurred but there was no tortuosity of the retinal veins.

Four months after operation, vision was $\frac{6}{6}$ and the papilla was entirely restored to normal. The ptosis was less and there was increased ability to turn the eye inward. There was a slight degree of enophthalmus.

One year later the enophthalmus was still present but there was very little ptosis. The right eye deviated outward and upward, behind a screen; when the right eye was covered the left eye fixed. Double images showed partial paralysis of the right superior rectus and the right inferior oblique. Vision was normal in each eye. There had been no pain or headache. There was no evidence of any recurrence of the growth.

Anatomical Findings.

Macroscopic.—The tumor apparently about half again the size of the eyeball was grayish white in color, soft and lobulated with a thin connective tissue capsule. On section, the cut surface was homogeneously translucent and gelatinous.

The growth after bisection was fixed in 10 percent formalin, dehydrated in the alcohols and imbedded in celloidin; sections were stained in hemotoxylin-eosin and in a simple hemotoxylin stain for mucin.

Microscopic Findings.—The intercellular substance appears finely granular with many small clear vacuolated spaces. The essential cells are star and spindle shaped with thin short processes extending from the pointed ends of the cell body. There are also many approximately round cells showing no processes. The nuclei of all these cells stain moderately dark with a fine grained chromatin

stippling; in shape each nucleus roughly approximates the shape of the cell body to which it belongs. Scattered diffusely among these essential cells are a few small lymphocytes. The relative frequency of essential cells varies greatly in different portions; there are more present where the blood-vessels are thicker. The blood-vessels are mostly small veins but with some small arteries and a few capillaries. Polymorphonuclear leucocytes are seen among the red corpuscles in the lumina of many of the blood-vessels, particularly around the periphery of the blood-stream. The special stain for mucin colors the grains and fine granules in the intercellular matrix a very light purple.

Sections were prepared at the Eye Research Bureau maintained by the Missouri Association for the Blind and the St. Louis University School of Medicine.

REPORT OF THE SECTION OF OPHTHALMOLOGY, NEW YORK ACADEMY OF MEDICINE.

By Dr. THOMAS H. JOHNSON, Secretary.

MEETING OF JANUARY 16TH, 1928. DR. BERNARD SAMUELS, CHAIRMAN.

Dr. Herbert Wootton's report of a case of **glioma of the optic nerve** will be published in full.

Discussion: Dr. Bernard Samuels: In 20 years there has been but the one case of this condition on record in the Laboratory at the New York Eye and Ear Infirmary.

Dr. Martin Cohen: In 1916 I described a primary intradural tumor of the optic nerve. The mass was an epiblastic mass similar to that shown by Dr. Wootton. I believe the cells are typically ganglion cells, and belong to the group that Ewing has described. The cells represent a nest of embryonal cells indicating a link between the retina and the brain. We could, therefore, diagnose the tumor as a ganglionic neuroglioma, according to Ewing. The ganglion cells can be recognized by special neural tissue stains. Unless the proper staining methods are used, it is difficult to say whether they are ganglion cells or not.

Dr. Arnold Knapp: Did the cut section of the optic nerve at the apex of the orbit show involvement, indicating a probable extension of the optic nerve tumor? In the tumors of the optic nerve which I have operated upon, the optic nerve at the apex of the orbit has been found involved, and the optic canal was enlarged. At the same time, in none of the cases has an intracranial extension become noticeable, though one case has now been observed for twelve years. In the "Contributions to Ophthalmic Science" dedicated to Dr. Edward Jackson (On the Intracranial Extension of Optic

Nerve Tumors) I drew attention to the importance of determining a possible intracranial extension of these tumors, and the earliest change which can be recognized is the X-ray demonstration of an extension of the sella under the anterior clinoid processes.

DR. WOOTTON: The X-ray showed enlargement of the optic foramen. In this seventeen-months old child, it was remarkable how well this showed up by X-ray. The pituitary fossa was normal.

DR. DAVID WEXLER presented a case of **chorioretinal depigmentation accompanying generalized vitiligo** which will be published in full.

DISCUSSION: DR. HELLER: Is vitiligo a common skin disease? Does it always occur in conjunction with these eye changes?

DR. WEXLER: It is considered a frequent disease by skin specialists, but generalized loss of pigment is not so common. It is more usual in the colored race. The American literature contains no record of eye changes of this type in association with generalized vitiligo. There are three or four instances recorded in the German literature.

ISSAC M. HELLER reported a case of **bilateral choked disc due to arsenic poisoning.** He said a girl 9½ years of age was seen by him on December 3, 1927, complaining of headaches for which he could find no cause. Vision, R: $\frac{10}{10}$, L: $\frac{10}{10}$. Nothing unusual was seen in the fundi. On December 27, 1927, the patient was again seen, and the vision with correction was R: $\frac{10}{10}$; L: $\frac{10}{10}$. At that time, there were choked discs of about 4 diopters in the right and 3 in the left, with an edema which spread almost to the maculæ. X-ray of teeth and sinuses, urinary examination, neurological examination were negative. Examination for arsenic showed 2+ for the urine and feces, and 4+ for the blood. Vision on January 9, 1928, was $\frac{10}{10}$ in the right eye and $\frac{10}{10}$ in the left. The source of the arsenic was believed to be from an inferior grade of candy.

DR. AGATSTON asked if the elevation of the optic nerve had decreased.

DR. HELLER: Yes, by two diopters.

A case of **trichinosis with exophthalmos** was reported by

DR. BEN WITT KEY in which there was found the characteristic symptom of œdema of the face and lids; a high eosinophilia of 69%; a temperature ranging to 104.5°F.; profound weakness, and irritability of the heart. The embryo trichinella found in a section of the deltoid muscle was shown in a microphotograph. The patient recovered after 17 days in the hospital.

DISCUSSION: DR. WALTER LESTER CARR: I had the opportunity of seeing the patient whose history has been given so fully by Dr. Key. There was a decided impression made by the edema of the upper portion of the face, eyelids and conjunctivæ. Prostration was severe. The pulse was quickened and weak, with a blood pressure of 110–70. The muscle tone of the heart was poor with an intensified valve action. The lungs showed a few large mucous rales. In view of the blood picture, I ordered thymol, colonic irrigations and saline aperients, and suggested neosalvarsan. As the parasites migrated into the tissue, there was an alteration in the percentage of eosinophiles and although the treatment was somewhat empirical, the result, as shown in the improvement of the patient, was satisfactory. I would emphasize the impression made by the edema of the face and eyelids, and also the prostration, as being more decided than in sinusitis. The diagnosis was completed by the blood examination, and the presence of the trichinæ in the muscle tissue.

DR. DANIEL B. KIRBY read a paper on **a modified Motais operation for ptosis,** illustrated with lantern slides, which will be published in full.

DISCUSSION: DR. WHEELER: Dr. Kirby has given me credit for having operated on seven of the twelve cases reported. Very wisely, Dr. Kirby has avoided discussion of the relative merits of the different types of procedures for the correction of ptosis of the upper lid. It may not be out of place for me to say, however, that one must put high value on the Motais idea. Dr. Kirby has demonstrated the feasibility of performing the Motais type of operation through a skin incision, without making an opening in the conjunctiva. This in itself is worth while. I should go so far as to say that of the various suggestions for operable ptosis, this may be the best if the ptosis is complete, and the power of elevation

of the globe present. Sufficient correction can be obtained, without persistent lagophthalmos, and without angulation of the upper lid region.

DR. JAMES WILSON CASSELL gave his observations of the Eye Clinic of Dr. Henry T. Holland at Shikarpur, India, with lantern slides and motion pictures, which will be published in full.

DR. SIGMUND A. AGATSTON then read a paper on **the fundus as a definite index to arterial disease** which will be published in full.

DISCUSSION: DR. HARLOW BROOKS: This paper has definite application to the work of the internist and it is of tremendous importance. Some do not sufficiently appreciate how valuable it is to us to receive from the eye men a detailed study of the condition of the retinal vessels. We internists also need your assistance in cases of this specific distribution of arterial disease, even though it limits itself to a specific field,—the vessels of the brain and the ophthalmic group of vessels. This does not mean necessarily, however, that the vessels of the kidney or heart are also diseased. A recent study of coronary disease in relation to the changes in the retinal vessels has given very discordant results. Angina pectoris also is not always due to coronary disease and can be also due to disease of the heart muscle, of the aorta or even the pericardium. We must not generalize too much. A collection of such statistics as Dr. Agatston has presented will give us a more correct viewpoint in relation to many obscure symptoms of the brain and cord in particular. We must be very cautious in assuming that high blood pressure and nitrogen retention are definitely related. Clinical study seems to show that they are not closely related, even usually. I wish that I could accept such a simple explanation as that essential hypertension is arterial spasm. That does not explain the complex picture that we see in our work. I think the retinal arteries are frequently the first to show changes where generalized arterial disease is present or is developing. These facts are frequently of value to the neurologist, and the internist, but must not be over-estimated as to accuracy. There is another method of reaching the same point for the internist not associated with an ophthalmologist, that is the study of

the conjunctival vessels, as brought out by Dr. Dennis, who is also an ophthalmologist. He used an ordinary hand lens with refracted light. We can certainly say that most of the arterial changes shown in the retinal arteries are also closely approximated by disease of the conjunctival vessels. The retinal and conjunctival vessels come from the same roots. Arteriosclerosis is not always associated with changes in tension. It is sometimes present with hypo- and not hypertension. Obstruction or thrombosis is not excluded by any means in cases of neutral tension. We must take these conclusions with much salt, but the work is very valuable. I hope the eye men will stand by and help the internists, but do not let us be too quick to jump to conclusions. The subject is a very large one and people who have studied it seem to be most convinced that they know very little about the problem as yet.

Dr. Boas: Dr. Agatston pointed out in his paper that the degree of blood pressure can be estimated by the degree of sclerosis of the arteries. It would be more logical to reverse that order and say that the degree of sclerosis of the retinal vessels is in proportion to the blood pressure. It is a very complex subject. Dr. Brooks pointed out the lack of correspondence of general arterio-sclerosis to change in the retinal vessels. Another point may be mentioned, and that is that we must distinguish between arterio-sclerosis of the larger arteries, and arteriolo-sclerosis. The kidneys, and also the retinal vessels are affected by the latter process. This explains the correspondence between the retinal and the kidney lesions. Patients with what was formerly called chronic interstitial nephritis are primarily cases of hypertension. Secondary disease of the arterioles occurs in many areas of the body, but most frequently in the spleen, kidney, pancreas and the retina. In the kidney it produces contraction of the kidney, or granular kidney. The correspondence of changes is not absolute. Many patients with hypertension have normal kidney function and normal retinal vessels. In general it seems to me that the degree of retinal arterial change corresponds to the diastolic pressure, rather than to the systolic pressure. The diastolic pressure is most important because it indicates the load carried by the heart and arteries

and corresponds more closely to the progress of the lesion. There is most change in the retina in the cases of sub-acute and chronic nephritis, where the lesion lasts many years. In addition to the height and duration of diastolic pressure, the rapidity of the course of the disease is an important factor. The most valuable result I expect of this study is the emphasis of the physiological importance of primary arterial hypertension, with secondary retinal changes.

MEETING OF FEBRUARY 20TH, 1928. DR. BERNARD SAMUELS, CHAIRMAN.

DR. RALPH I. LLOYD and DR. PAUL COLLINS presented four cases:

CASE 1. **Synchisis scintillans** of the **anterior chamber.**

The patient, aged 30, lost the sight of the left eye after an injury at the age of 6. The blind eye was recently struck and has been injected and tender. On the anterior surface of the iris there are hundreds of minute dots that shine like tiny spangles; they are fixed and do not move, and seem to be held in an invisible membrane. The slit-lamp shows these shining dots as quadrilateral crystals as bright as any mirror. This condition has been observed in two types of cases; in one, the crystals originate in the posterior chamber and pass into the anterior chamber. They come from an over-ripe ruptured lens or from the vitreous with an intact lens. The second group has crystals originating in the anterior chamber apparently from an organized exudate in the angle of the chamber. About 60 of these anterior chamber collections of cholesterin crystals have been reported.

CASE 2. **Tumor of lachrymal gland** of 30 years' duration.

The patient died a short time before the meeting, of an abdominal malignancy which was asserted to be a metastasis from the lachrymal gland, but this hardly seems probable. In 1927, two attempts were made to remove the lacrymal tumor but the patient collapsed under the anæsthetic and the operation was abandoned. In 1906, a large mass was removed that looked like "boiled hominy with threads." X-ray treatments were then given for several years. (Lantern Slides of the patient show the huge mass pushing the eye downward and forward.) The tumor is probably a "dacryops."

CASE 3. **Bi-lateral iridocyclitis** (tubercular) precipitated by mild trauma.

The patient, a young colored girl of 20, was struck on the left eye by a basket-ball. In a short time, this eye became sensitive to light but there was very moderate circumciliary injection. At this time typical nodules were present on the iris edge and upon the anterior iris surface about midway from pupil margin. She attends at the Clinic only at long intervals, yet there are very few adhesions in either eye. No nodules are now visible but the posterior corneal deposits persist. The vision is but little affected.

CASE 4. **Nodular iritis** in a colored man with a positive history of syphilis. The iris is adherent, there are two nodules on the iris surface about midway from pupillary edge to margin, one at 10 o'clock and the other at 4 o'clock. They are larger than the nodules of the preceding case, whiter and seem to be in the iris tissue, and only exposed to view at the tip of the mass. The eye is red and painful.

DR. LEWIS W. CRIGLER presented a colored drawing of **haloes in glaucoma** as seen and drawn by a patient in an attack. The characteristic glaucomatous halo was very clearly depicted with the predominating colors of blue and red in their true positions. The speaker spoke of the importance of this symptom as a diagnostic aid, in the fleeting attacks which occur in the early stage of the disease.

Dr. Crigler presented a patient, male, 58 years of age, with **retinitis pigmentosa,** upon whom he had operated in 1915, removing a posterior cortical cataract from his right eye. His vision before the operation was $\frac{1}{200}$ and field contracted to within 10° of the fixation point. Two months after operation vision was improved to $\frac{20}{40}$.

Patient has been continuously occupied as a druggist, until two months ago, when it was found necessary to needle the eye on account of a thin membrane. He is back at work, can read signs across the street, and the finest print with proper lenses, notwithstanding the fact that he has nothing but macular vision left.

DR. CRIGLER presented a case of **couching for cataract.**

F. L., aged 56, a colored physician, a native of Liberia (educated in the United States), four years ago developed

cataracts while practising among the natives of his country. In 1924, an itinerant doctor couched the left lens. The immediate result was very gratifying. Two days later pain and inflammation set in. The eyeball became red and the pain was excruciating. Acute symptoms persisted for several weeks, and the eye was not entirely quiet for one year, during which time he was unable to work. There has been no recurrence of any trouble in the eye for the past three years.

The shrunken lens can be seen behind the iris in the infranasal quadrant, resting on the ciliary body. The pupillary space is perfectly clear, and the fundus is normal. Vision, with correcting lenses is $\frac{1}{10}$ and Jaeger type No. 1 can be read with ease. He comes from Africa to this country to have the right eye operated on in accordance with present methods.

DISCUSSION: DR. ARNOLD KNAPP, I think we should be very cautious in interpreting what patients describe as haloes. In order to clarify this, Colonel Elliot suggested some years ago a contrivance consisting in a thin layer of lycopodium between two pieces of glass. If this is held up in front of a light, a true spectrum is observed.

The favorable result that Dr. Crigler has obtained in his case of retinitis pigmentosa is another evidence of the value of this operation. I would suggest as a counter-indication, very poor vision. In other words, if the vision is below $\frac{20}{200}$, the result will not be anywhere near as favorable as when the vision is better. These eyes recover perfectly well from the cataract operation, though the recovery is somewhat delayed because of the slow absorption of the clear cortex unless the lens has been removed in the capsule.

Dr. Crigler's third case is not technically an example of a lens-couching, as I think that term should be kept for cases in which the lens is displaced into the vitreous. In this particular case, the lens has been displaced laterally, so that it still lies in its former plane but behind the iris, where apparently it has caused less damage and has led to no degeneration of the eye, as is customary after the couching operation.

DR. KAUFMAN SCHLIVEK showed a case of **gumma of the orbit.**

The patient, a widow aged 39 years, was seen on Novem-

ber 23, 1927. Vision, right eye, had always been poor, and blind for 10 years. Ten days ago the right eye became red followed by sharp pain and throbbing over the right side of the head three days ago; there was also nausea and vomiting. For one day, there has been a rapidly increasing swelling about the right eye.

Examination: O.D.—No L.P. O.S.—$\frac{1}{200}$, myopia, with correction $\frac{1}{1\frac{5}{8}}$. There was enormous edema of both lids and very marked chemosis. The upper two-thirds of the cornea was covered by this firm edematous conjunctiva. The lachrymal sac was negative. No pain or tenderness on pressure over the globe. The eye was in extreme proptosis, and absolutely fixed. There was no bruit or pulsation, and the exophthalmos could not be reduced. The cornea showed deep infiltration, close to the limbus and of the ring type. Palpation for tension was impossible. Cornea was anesthetic, but there was no anesthesia in the area supplied by the first division of the trigeminal nerve. The lower lid left eye showed slight edema. T.P.R. normal.

The first diagnosis was orbital abscess. The most common cause is accessory sinus disease. The eye was pushed directly forward. Even in marked cases of orbital abscess due to sinus disease, the proptosis is generally greater in one direction. X-rays were negative. There was no local or metastatic infection and no trauma. No pain, tenderness, or temperature. The next diagnosis was tumor—the condition was too acute. The picture really looked like cavernous sinus thrombosis, but the patient wasn't ill. The condition could only be the so-called inflammatory tumor, gumma or tubercle.

The patient was admitted to the N. Y. E. & E. Inf. the same day. The orbit was explored with a wide gauge needle. No pus was obtained; the fluid consisted of serum and a few leucocytes. Wassermann—four plus. Urine; negative; blood-sugar: normal. X-ray, Nov. 23: slight cloudiness of left ethmoids and sphenoids, otherwise negative.

Treatment consisted of bismogenol. This was repeated in four days, and very promptly the condition showed improvement. Neoarsphenamine Hg inunctions and large doses of iodides were given. The improvement continued but the Wassermann still remained strongly positive. The edema

chemosis and proptosis completely disappeared. There is no interference with eye motility. There is some injection of the conjunctiva. The cornea is opaque, and has a vascularized area. The epithelium has broken several times, but the eye is quiet. The cornea is small and the eye somewhat shrunken.

DR. JOSEPH J. ELLER showed a case of **button-like epithelioma involving entire right upper eyelid, cured by supersoft (2A) Roentgen rays.**

The patient, C. B., was a male 45 years of age, who was referred to the Dermatological Department of the Cornell Clinic by Dr. Bernard Samuels. He presented a lesion which involved the entire right upper eyelid. On palpation the lesion felt cartilaginous, almost button-like. There was some crusting.

The eyelashes were present. The weight of the growth was such that the eyelid drooped, covering almost the entire eyeball. This growth had been present for 14 years. It started as a pin-head sized nodule, and gradually grew to the size which the patient present at the examination. The lesion had never been treated. The patient was seen in consultation by various dermatologists, all of whom agreed with the diagnosis of basal cell epithelioma.

The lesion was given six treatments with the so-called supersoft Roentgen rays, which are rays of extremely long wavelengths, having in their beam a large percentage of X-rays between $\frac{1}{8}$ and 2A in wave length. The penetration of these wave lengths is comparatively slight as not more than 5% of the incident radiation reaches the level of the hair follicles. This radiation is produced by a specially constructed hot cathode tube built on the same principle as the Coolidge tube, with the addition of a Lindemann glass window which permits the long waves to pass through, which the ordinary glass of the usual Roentgen ray tubes does not. Each of the doses given for the first four treatments was twice the erythema. The (last 2) treatments were one erythema dose each, the intervals were 2 to 4 weeks. The first treatment was given February 10, and the fourth treatment was given in June, 1927. The last treatment was given July 15, 1927, and there has been no treatment since.

The voltage used was 8,000 and the distance 6*cm*. No protection was given the eyeball although the skin was screened outside of the involved area. The eyelashes never fell out at any time. At no time did the patient complain of any symptoms in that eyeball. Two ophthalmologists, Dr. Bernard Samuels and Dr. Martin Cohen examined the eyeball at intervals. They found no evidence of inflammation at any time. The patient is now presented in order to show the result of treatment. On it can be seen the lesion has entirely disappeared, the eyelashes are intact, and the skin is soft and nonatrophic.

DR. GEORGE H. SEMKEN'S paper on **eye and orbit phase of cancer work at the New York Skin and Cancer Hospital** is to be published in full.

REPORT OF THE TRANSACTIONS OF THE OPHTHALMOLOGICAL SECTION OF THE ROYAL SOCIETY OF MEDICINE.

By Mr. H. DICKINSON, London.

A clinical meeting of the Section took place on March 9th, at St. Thomas' Hospital, the President, Mr. Ernest Clarke, C.V.O., occupying the chair.

Mr. P. G. Doyne showed a case of

Plagiocephaly.

The skiagram showed a definite thickening of bone, and the right eye was hypermetropic, suggesting a smaller orbit than that on the other side. There seemed to be nothing else abnormal about the patient.

He also showed a case of **Parinaud's conjunctivitis.** He received a blow on the eye from another boy, and since then the eye had been inflamed and swollen. X-ray and bacteriological examinations were negative.

Mr. E. R. Chambers mentioned a similar case to the foregoing, in which he used 2% boric acid and irradiation with ultra-violet light to the whole body. In two months the condition had cleared up.

Mr. A. S. MacCallan showed a man, aged 36, with

Tumor of the Iris.

He asked for help in regard to the exact diagnosis. The patient's fundi were normal. The slit-lamp showed the tissue of the de-colorized tumefied area merged from the iris into a de-colorized mass. His own view was that it was a nævoid growth of the iris; it did not appear to be a cyst.

Mr. Humphry Neame agreed it was probably nævoid. He suggested that an accurate measurement of it be taken now and again in a few months time, as probably it was increasing, and he thought it would ultimately prove to be malignant. If it did enlarge, he recommended operation; he was not sure whether it could be got away by means of iridectomy.

Mr. Cyril Walker referred to a case in a boy aged 16, who had a lump similar to that in the case now shown. It grew slowly, and the speaker did iridectomy. There was very little pigmentation. For a month or two after that operation there was no sign of it, but after six months there was a distinct recurrence in the angle. Mr. Treacher Collins, who saw the boy, said it was clinically sarcoma. The eye was excised. The patient was still well, 11 years afterwards.

Mr. Basil Graves thought the growth was very superficial and cellular, and that it was a nævoid growth.

Mr. A. Hugh Thompson showed a patient with

Aniridia and a Dislocated Lens.

The father of the patient appeared to have been affected in the same way. Each lens was dislocated upwards; no iris could be seen.

Dr. G. W. Kendall exhibited a patient, a female, who had loss of the upper half of the visual field of one eye, but visual acuity was $\frac{6}{9}$ in each eye. There appeared to be an obstruction of the inferior retinal arteries.

Partial Avulsion of Optic Nerve.

Mr. A. D. Griffith showed a boy, aged 18, who presented, in his left eye, a mass of scar tissue around the optic disc. The picture was that of partial avulsion of the optic nerve, and it might have been due, he thought, to a birth injury.

Mr. Basil Graves spoke of seeing a similar condition resulting from a blow with a pitchfork. In the present case there was probably some trauma.

Mr. A. Hugh Thompson exhibited a patient with acquired central choroiditis simulating a second optic disc. A year

ago, when he first saw the man, the right eye was in much the same condition as now, i.e., with a central patch simulating the optic disc. There was only peripheral vision. Last November the patient came again and stated that his left eye was going wrong. His vision was slowly deteriorating, and last time it was examined it was only $\frac{6}{18}$. There was a distinct patch in the macular region, probably, he thought, an early stage of the same condition as that in the right eye. It was probably a macular degeneration. The man was a heavy smoker; three of his teeth had been extracted.

Mr. H. Neame's view was that it looked like a central senile exudative retinitis.

Mr. Davenport felt no doubt that this came into the class of senile exudative retinitis, and he thought the patient showed cardio-vascular, or at least vascular, degeneration.

Mr. Hudson showed a case of

Microphthalmos due to Intra-uterine Inflammation.

He said it seemed to be a definite example of ulceration of both corneæ before birth.

NOTICE.

Invitation to Ophthalmologists to attend the International Ophthalmological Congress to be Held in September, 1929, in Amsterdam and The Hague (Holland).

In the name of the International Council and of the National Committee we have the honor to invite you to take part in the International Ophthalmological Congress which will be held in Holland in September, 1929.

We beg to point out that members intending to read any paper at the Congress should, prior to the first of June, 1929, address an abstract, containing the principal conclusions, to the Secretary, Prof. Dr. Zeeman.

No communication will be admitted on any subject, unless the respective abstract has been received by Prof. Zeeman not later than the first of June, 1929.

The subscription amounts to Dutch Fl. 25.

We request you to apply for membership as early as possible to the Secretary of the National Committee: Prof. Dr. W. P. C. Zeeman, Wilhelmina Hospital, Amsterdam, Holland. Please remit your subscription to the Treasurer, Dr. H. M. Roelofsz, Managing Director of the Incassobank, 531 Heerengracht, Amsterdam.

Further communications concerning the meetings, symposia, accommodations, excursions, etc., will be addressed to you in due course.

For the International Council:
J. Van Der Hoeve, *Chairman.*
E. Marx, *Secretary.*

For the National Committee:
J. Van Der Hoeve, *Chairman.*
W. P. C. Zeeman, *Secretary.*

Coöperators (in alphabetic order) of the International Congress of Ophthalmology, to be held in Amsterdam and The Hague, Sept. 5–13. 1929.

Symposium	*Rapporteur*	*Domicile*
1. Geographical expansion and social international campaign against trachoma.	Arganaraz	Buenos Ayres
	Gronholm	Helsingfors
	von Gross	Budapest
	Maggiore	Sassari
Dr. Wibaut of Amsterdam will present and discuss the "mappa mundi" about the expansion of trachoma.	Mijashita	Tokio
	Sobhy Bey	Çairo
	Soria	Barcelona
2. Etiology and non-operative treatment of glaucoma.	Duke Elder	London
	Hagen	Oslo
	Magitot	Paris
	Wessely	Munich
3. Diagnosis of supra-sellar tumors.	Christiansen	Copenhagen
	Cushing	Boston
	Holmes	London
	Van Bogaert	Antwerp

Moreover the following reports will be presented to the International Ophthalmological Council, which will publish and send these reports to the members of the Congress.

Subject	*Rapporteur*	*Domicile*
a. Examination of visual acuity.	Dufour	Nancy
	Elschnig	Prague
b. Perimetry.	Lauber	Vienna
	Peter	Philadelphia
	Traquair	Edinburgh
c. Notation of the cylinder axis.	Marquez	Madrid
	Nordenson	Upsala
d. Examination of light sense.	Hertel	Leipsic
	Ovio	Padua
e. Standardization of the visual examination of railwaymen, motor drivers, seamen and airmen.	Engelking	Freiburg i/Br.
	McMullen	London
	Onfray	Paris
	Verrey	Lausanne
f. Uniformity in the program of ophthalmological studies.	Lindner	Vienna
	Parker	Detroit